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गुरुकुल कांगड़ी विश्वविद्यालय, हरिद्वार
पुस्तकालय

विषय संख्या 610.7
पुस्तक संख्या M14 A
आगत पञ्जिका संख्या 46,540 E

पुस्तक पर किसी प्रकार का निशान लगाना वर्जित है। कृपया १५ दिन से अधिक समय तक पुस्तक अपने पास न रखें।

पुस्तकालय
गुरुकुल कांगड़ी

स्टाक प्रमाणीकरण १९८४-१९८५

46,540 E

An Abstract of the Proceedings

of

The Academy of Indian Medicine

MADRAS

(From 1st April 1941 to 31st Jan. 1942.)

CHECKED 1973
Initial

पुस्तकालय
गुरुकुल कांगड़ी

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ऋते ज्ञानान्न मुक्तिः
पुस्तक सं. 610.7
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दिनांक 46,540
गुरुकुल पुस्तकालय काँगड़ी

AN ABSTRACT OF THE PROCEEDINGS OF THE ACADEMY OF INDIAN MEDICINE

(*From 1st April 1941 to 31st January 1942.*)

During the period between 1st April 1941 to 31st January 1942 the Council of the Academy of Indian Medicine met five times under the Presidentship of Vaidyaratna Capt. G. Srinivasamurti, B.A., B.L., M.B. & C.M., to transact business relating mainly to organisational work including the enrolment of the 75 Foundation Fellows as per Clause II of the Constitution of the Academy (Vide Appendix A). So far, there are 60 Foundation Fellows including the Office Bearers and Members of the Council (Vide Appendix B). The Council also enrolled Honorary Fellows as per Section 4 of Clause V of the Constitution of the Academy (Vide Appendix B).

Certain schemes for research as shown in Appendix C, were approved and the following persons were appointed as Honorary Directors to direct the researches :—

1. Diagnosis and Prognosis by Examination of Urine by methods of Indian Medicine.

Rao Bahadur Dr. M. R. Guruswami Mudaliar, B.A., M.D.	
Ayurvedacharya Sri N. Madhava Menon	(Ayruveda).
Sri V. Narayanaswami Iyer, H.P.I.M.	do
,, C. N. Kuppuswami Mudaliar, H.P.I.M.	(Siddha).
,, K. Kasinathan, H.P.I.M.	do
,, M. Vadivelu Mudaliar	do

2. Indian Surgery : Treatment of Vrana.

Sri M. Parankusam, L.M. & S., F.I.M.
Rao Bahadur Dr. T. S. Tirumurti, B.A., M.B.C.M., D.T.M. & H.
Ayruvedacharya Sri N. Madhava Menon.
Sri I. V. Sharma.
Ayurveda Visarada M. K. Vaidyar, A.M.A.C.

In the first Council meeting, Sri C. Dwarakanath, L.I.M., (Madras) Z.T., (Hamburg 'Varsity) presented a paper on the problems of Tuberculosis and its treatment, based upon work done in the hospital attached to the School of Indian Medicine. (Vide, transactions of the Academy of Indian Medicine, Issue No. 1 published separately). This work will be continued on an enhanced scale in the near future.

With regard to item 1 of Research work noted above, preliminary investigations on the possibility of following the oil when dropped on liquids in general and urine in particular with Cine-Photography were done, by the Research Scholars Messrs. C. B. Hanumantha Rau, B.SC., L.I.M., and Y. Kondal Rao, L.I.M. The results they were able to obtain have been promising (Vide Appendix C). Photographic part of the investigation had to be temporarily stopped on account of the scarcity of Cine films due to the war conditions. Further work on this subject will be continued during the pendency of war without photographic work.

The research scheme on Vranam by Sri. M. Ramanatha Kamth has not been proceeded with, for want of a special out patients' and in-patients' surgery clinic. This will also be taken up at an early date.

SCIENTIFIC MEETINGS

Three Scientific meetings took place during this period, (Vide Appendix A) when papers on 'Abdominal pain in relation to food' based on the experiences and observations made in the hospital attached to the School of Indian Medicine was read by Sri V. Narayanaswami Iyer, H.P.I.M., (Vide Transactions of the Academy of Indian Medicine—Issue No. 2 published separately). This was followed by a discussion in which Doctors Syed Tajudin, Nazeer Ahmed Sayeed and the President participated.

Khan Bahadur Dr. Syed Tajudin, one of the Foundation Fellows, read an interesting paper dealing with the subject mentioned above, from the allopathic stand point of view. (Vide Transactions of the Academy of Indian Medicine Issue No. 3 published separately).

'Comparative Study of Varthma, Sitha and Sandhi Rogas' formed the subject matter of a paper read by Sri M. A. Nair, M.B. B.S., D.O., (Oxen), D.O.M.S. (Lond.) Zeug. Oph. (Vienna) which will be published shortly.

WEXFORD HOUSE
KILPAUK, MADRAS
15th February 1942.

C. DWARAKANATH,
V. NARAYANASWAMI,
Joint Secretaries.

APPENDIX A

MEETINGS OF THE COUNCIL.

	Date.	Subject.	President.
1.	5-5-1941.	Business Meeting and a paper on Tuberculosis was read by Sri C. Dwarakanath, L.I.M., Z. T., (HAMBURG 'VARSITY)	Vaidyaratna Capt. G. Srinivasamurtii B.A., B.L., M.B.C.M.
2.	29-6-'41.	1. Admission of Foundation Fellows. 2. A scheme for research in Urine was submitted and considered.	...
3.	31-7-'41.	1. Admission of Foundation Fellows, 2. Appointment of Honorary Directors for Research in Urine and vranas considered.	...
4.	21-9-'41.	Election of Foundation Fellows and Honorary Fellows.	...
5.	12-1-'42.	Admission of Foundation Fellows and to consider printing transactions of the Academy.	...

Scientific Meetings.

1.	20-10-'41.	Abdominal pain in relation to Food by Sri V. Narayanaswami, H.P. I.M.	...
2.	26-11-'41.	Modern views on abdominal pain in relation to food by Dr. Syed Tajudin Sahib Bahadur, L.M.P.	...
3.	29-1-'42.	Comparative study of Varthma, Sitha, and Sandhi Rogas (affections of Lid and conjunctiva) by Sri M. A. Nair, M.B. B.S., D.O., (Oxen), D.O. M.S., (Lond.), Z.O. (Vienna).	...

APPENDIX B

The Academy of Indian Medicine, Madras

OFFICE BEARERS

President :—

Vaidyaratna Capt. G. Srinivasamurti, B.A , B.L., M.B. & C.M.,
Latice Bridge Road, Adayar, Madras.

Vice-Presidents:—

Rao Bahadur Dr. M. R. Guruswami Mudaliar, B.A., M.D.,
Chetpet, Madras.

Ayurvedacharya Sri N. Madhava Menon, A.M., A.C.,
Malabar Ayurvedic Pharmacy,
Broadway, Madras.

Joint Secretaries :—

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Z.T. (Hamburg ' Varsity),
' Wexford House ', 75-76 Poonamallee High Road,
Kilpauk, Madras.

Sri V. Narayanaswami, H.P.I.M.,
Government School of Indian Medicine,
Kilpauk, Madras.

Treasurer :—

Sri Raja Dinakar, L.I.M.,
' Appar Agham ',
Royapettah High Road,
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Members of the Council :—

Sri M. A. Nair, M.B.B.S., D.O. (Oxen),
D.O.M.S. (Lond.) Z.O. (Vienna),
Government School of India Medicine,
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Government School of Indian Medicine,
Kilpauk. Madras.

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Government School of Indian Medicine,
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12 Amirunnisa Begum St.,
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Sri M. Koteswara Rao, L.I.M.,
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Atreyapuram, East Godavari Dt.

Sri A. Arunachalam, L.I.M.,
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Sri M. Mallikarjuna Rao, L.I.M.,
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Kalpataru Palace, 223, Chittaranjan
Avenue North, Calcutta.

Kavi Vinodh, Vaidya Bhushan Pandit Thakur Datta Sharma
Vaidya, Amritdhara Pharmacy,
Amritdhara Buildings, Lahore.

Vaidyarathna Pandit Rama Prasad Vaidya,
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'P. Subbaroya's Ayurvedic and Siddha
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Vaidyasastranipuna,
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Opthalmic Surgeon, Trivandrum,

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'Ayurveda Vidwan,' Sri Vithal Dispensary,
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Taj Pharmacy,
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Superintendent, Ayurvedic Hospitals,
Trichur, Cochin State.

Ayurvedacharya Ram Raksha Pathak, G.A.M.S., (Behar),
~~Medical Officer-in-charge,~~ Principal
~~District Board Ayurvedic Dispensary,~~ Ayurvedic Colle
~~Ekma Post, Saran District (Be~~har).
Gurukul University,
Gurukul Kangri
Hardwar

Sri V. T. Jagadeeswaran, L.I.M.,
~~Medic~~al Officer,
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L. F. Siddha Dispensary,
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Dr. Balakrishna Pandurang Delwe, M.D., Z.I.P. (Wien),
Children and Eye Specialist,
Itwari, Nagpur City, C.P.

APPENDIX C

A scheme of research on the examination of urine according to Indian systems of medicine to assess diognosis and prognosis of diseases

BY

C. B. HANUMANTHA RAU, B.Sc., L.I.M.

and

Y. KONDALA RAO, L.I.M.

The following will be in brief the scheme of work on the "Investigation on the diagnostic and prognostic value of the examination of urine according to Indian systems of medicine (Siddha, Ayurveda and Unani)". In doing so, we have tried to get an almost true translation of the original works on the subject from Indian medicine. The inspiration for this work has come from Rao Bahadur Dr. M. R. Guruswami Mudaliar, B.A., M.D., who suggested in his Presidential Address of the 10th L.I.M. conference, that the oil test employed to examine urine by practitioners of Indian medicine has great scientific value in assessing the diagnosis and prognosis of diseases. This test could be studied rationally with the help of cine-photographs and other modern appliances.

This research when successfully completed may offer an easy and simple method of examination of urine for the determination of diagnosis and prognosis of diseases. This may, while establishing the scientific basis of the easy and simple methods available in Indian medicine, also contribute something new and useful to modern medicine.

The problem of urine examination according to Indian systems of of medicine has two distinct aspects :

1. The appearance of different colours in different specimens of urine in reflected sun light.

2. The pattern assumed by a drop of sesamum oil in different samples of urine.

The gist of the whole problem will be as follows :—

A known quantity of a specimen of urine from a particular patient whose clinical diagnosis of the disease would be established and recorded both according to Western and Indian systems of medicine will be subjected to critical study, both on the Indian and Western side, by known methods. The details of the methods of investigation proposed to be employed will be recorded separately in tabular statement.

To make the research simpler, we propose to follow diseases in which there are corresponding tests of urine both qualitative and quantitive in western medicine, as for instance, in Diabetes, Albuminuria, Typhoid and Tuberculosis, with Fehling's Test, Nitric Acid Test, Diazo reaction and Diazo and Weiz reactions respectively. In the nature of things, it is extremely probable that by plain and simple tests enumerated in books on the subject in Indian medicine, sugar, acetone albumin, etc., may be qualitatively and quantatively determined. Though it is contrary to the spirit of science and scientific methods to anticipate conclusions in advance, we may be excused in this specific instance to expect a drop of oil to assume different patterns in different specimens of urine containing varying quantities of almost the same substance or substances. We may be permitted to illustrate this with percentage of sugar in urine. In one specimen of urine for instance, containing half a per cent. of sugar and in another specimen of urine containing three per cent. of sugar, the oil drop need not assume the same pattern or sink to the same depth. When this investigation is elaborated and applied to several specimens of urine containing identical substances in varying quantities, we may be right in anticipating a data which may help us in the qualitative and quantitative estimation of such substances. In other words, the oil drop may assume an exaggeration of the same pattern or assume a totally different pattern in a series of samples of solutions having a known substance in definite increasing dilusions. Hence the excuse.

With regard to the materials required and their approximate cost, the following table gives an idea :—

Materials.	*Approximate cost.*
1. Reagents :— Fehling's Soln, etc.,	A sum of Rs. 30 may be sanctioned to carry on the preliminary investigations.
2. Apparatus :— Flasks, test-tubes, etc.,	
3. Cine-Films,	
4. Stationery,	

The details of each item, the exact number and the cost will be submitted as soon as the preliminary laboratory investigation is finished.

In short, this is the scheme of work which we have proposed to take up under the auspices of The Academy of Indian Medicine.

Examination of urine according to Indian systems of medicine [Text with Translation of Therayar's Nir Nirakuri Neikuri]

By

C. B. HANUMANTHA RAU.

அருந்தும் ஆறிரதமும் அவிரோ தமதாய்
அஃகல் அலர்தல் அகாலவூண் தவிர்ந்தழற்
குற்றள வருந்தி உறங்கி வைகறை
ஆடிக் கலசத் தாவிஐ காதுபெய்
தொருமுகூர்த் தக்கலைக் குட்படு நீரின்
நிறக்குறி நெய்க்குறி நிருமித்தல் கடனே.

The patient is advised to take a well-balanced diet with compatible ingredients, within limits of his appetite, at the proper time. He is advised to have good sleep and then in the early hours of the following morning he has to void urine in a clean glass vessel. This urine is subjected to analysis within one *Muhurtham* i.e., 1½ hours (before it cools down or when it is still luke-warm).

அருப்ப முற்றார்க் கவ்விதி விலக்கே.

This rule does not apply to those who are acutely ill.

வந்தநீர்க் கரியெடை மணம்நுரை எஞ்சலென்
றைந்தியல் உளஅவை அறைகுது முறையே.

The general characteristics to be noted of urine are colour, relative density, odour, froth and quantity.

COLOUR

பீதம் செம்மைபை கருமை வெண்மையென்
றோதைங் கொழுமையை ஒத்துகு நீரே.

In colour the following are the varieties :— Yellow, Red, Green, Black and White.

அரிசனத் தாறும் அருணத்து நான்கும்
அரிதத் தைந்தும் அஞ்சனத் தொருநான்கும்
வெண்மையுள் இரண்டுமாய் விள்ளுஞ் சுருதியே.

Among these colours there are six shades in yellow, four in red, five in green, four in black and two in white.

YELLOW

மஞ்சள்நீர் விகற்ப வன்னம் புகலுதும்.

About the various shades of yellow colour ;

நீரணும் வழுதுகு நீத்தம் நிறமெனின்
சீரணக் குறைவின் அபக்குவ நீரே.

If the colour of urine resembles the yellow colour of rain water dripping from the edge of a thatched roofing or of the water in which straw is soaked, then it indicates indigestion.

பாரில் துரிஞ்சிப் பழநிறம் பொருந்தில்
சீரண பக்குவ நீரெனத் தேரே.

If the colour of urine resembles the colour of the peel of lime fruit, it indicates normal digestion.

செம்மையும் நிசியும் சேர்ந்தெழு நிறமெனின்
வெம்மை உயர விளைந்தவிர் நீரே.

If the colour of urine has a shade of red and yellow colour, it indicates excessive heat in the system.

காட்டின் நரந்தக் கனிநிறம் காணில்
சூட்டைத் தரற்குத் தொடுகுறி நீரே.

If the colour of urine resembles the yellow colour of the peel of wild *Narandhai* fruit, it indicates impending heat excess to a second degree.

கொதிஅனல் நிறத்தின் கொள்கையே யாயின்
அதிஉட் டினத்தை அடைந்தநீர் ஆமே.

If the colour of urine has a reddish yellow tint like the colour of flame, it indicates heat excess to a third degree.

பீதமுங் குங்குமப் பூவுமா ரெழிறரின்
எதிதன் மேல்வெப் பின்றெனு நீரே.

If the colour of urine is yellow with the colour of saffron, it indicates an extremely over heated system.

RED

செம்மைநீர் விகற்பச் செய்தி நவிற்றுவாம்.

About the varieties in red colour :—

அரத்தமுங் கருமையும் அண்ணிய கறைதரின்
இரத்தப் பொங்கலால் இறங்கித நீரே.

If the colour of urine is red with a shade of black, it indicates blood pressure (?) (Raktham Pongudhal).

செக்கச் சிவந்தபன் னீர்ப்பூப் புகருறின்
மிக்குதி ரக்கொதிப் பால்உகும் நீரே.

If the colour of urine is bright red like that of a red rose, it indicates blood pressure (?) of the second degree (Mikka udhira kodhippu).

கருமையுள் செம்மைசேர் கறையும் சிவப்பும்
குருதி பொங்கிய குறிபுகல் நீரே.

If the colour of urine is black with a shade of red or is extremely red, then it indicates the highest degree of blood pressure (?) (Kurudi Pongudhal).

GREEN

பச்சைநீர் விகற்பம் பன்னுது மிவணே.

About the varieties in green colour :—

கருமைச் சாமளக் கருவுறு மாயின்
வருசீ தளத்தால் வரப்படு நீரே.

If the colour of urine has a shade of black in its green, it indicates the impending origin of a state of coldness (Seethalam) in the system.

இடத்துறு குணத்தை யேய்க்கு மெனிலோ
விடத்துறு சீதளம் மேவிடு நீரே.

If the colour of urine is bluish green like the sky, it indicates a state of coldness as a result of poisons.

தகுமொரு நீலச் சாயைகண் டிடினே
மிகுசீ தளத்தால் விளைநீ ரிதனால்
மருத்துநோய் பால விருத்தர்க்கு மாமே.

If the colour of urine has a shade of blue, it indicates excessive coldness (Seethalam) in the system. It shows impending development of *Vatha* diseases both in young and in old.

அம்மநீ லத்தரு வார்ந்துந் தடிக்குமென்
மும்மலச் சிதைவை முளைப்பிக்கு நீரே.

If the urine is blue and also a little thick, it indicates the impending derangement of all the three *doshas.*

சொக்கிலைப் பச்சையின் தோற்ற முளதேல்
மிக்கு மும்மலம்கெட விழுநீர் கொடிதே.

If the colour of urine is bright green like that of a leaf, then it indicates the excessive provocation of all the three *doshas.*

BLACK

கருமைநீர் விகற்பம் கழறப் புகுவாம்.

About the varieties in black colour :—

வான்மீக வெழிற் குண்மைக் குணமுறின் அது
தான்கா மாலைநோய் தரும்அஃ தின்றெனில்
பித்தநோ யேனும் பிறக்கப் பணிக்குமே.

If the urine has a tinge of black in a saffron colour, it indicates the causation of jaundice or atleast the production of *Pitta* disease.

அம்மையுஞ் சிறிய செம்மையுஞ் சிவணினே
அரத்தத் தழிவுஎன்று அறைதரு நீரே.

If there is a tinge of red colour to the above mentioned colour of urine, then it indicates the vitiation of blood (Raktha Azhivu).

கார்க்குள் புன்னரிக் கருவிரவிய தெனின்
ஆர்க்கத்து அழுக்கை அறிவித்த நீரே.

If the urine is dark with a shade of green, it indicates impurities of Raktham.

மையும் வெளிறும் மருவிய சாயையேல்
ஐயுயிர் மிகக்கொதித்து அழிவுறு நீர்இது
காலா தீதக் கனல்பவத்து உடையது
மாலாந் தருண சுரத்தர் விருத்தர்க்கு
உண்டாம்ஐத் திரவியத்து ஊணால் கொடிதே.

If the urine is black with a shade of pale colour, it indicates an excessive provocation of *Sleshma* and *Vatha*. It is the result of a continuous state of very great excess of heat in the body. This is found in patients who are suffering from *Tharuna Jwaram* and also in old people. It also results after ingesting more of *Sleshma* provoking substances and it is of bad prognosis.

WHITE

வெண்மைநீர் விகற்பம் விரித்தினிது உரைப்பாம்.

Now about the varieties in white colour :—

வெண்மையு முற்று மிகத்தெளிவு உடைத்தேல்
உண்மையாஞ் சுத்த சீதளத்து உதகமாம்
இந்நீர் வசப்படாது இவனாய்யுந் தரம்
முந்நீர்ப் பெருக்கு அமிழ்வான் உய்தல் ஒக்குமே.

If the urine is clear and pale, it indicates a state of absolute coldness in the body. This is of bad prognosis.

அறவெளிர்ப்பினும் சளியைப்போல் விழின் அது
மறவன்அதி கொதிப் பால்வரு வனமே.

If the urine is pale or white like the expectorated sputum, it denotes excessive provocation of *kapha*.

அமிழ்து அரியெனில் என்புஅற வுருக்கியநீர்
அஃதே கயநோய் ஆக்கும் அந்தத்தே.

If the urine is milky in appearance, it indicates destruction of bone-marrow. It will ultimately lead to *kshayam*.

காணிதில் சீழுங்கலந்து இழிமணம் உறின்
கருப்பநாபிகள் உளுங் காமநா ளத்துளும்
விரணமுண்டு இன்றேல் எய்தும் அஸ்மரியலது
இருத்தலே திண்ணம் எனமனத்து எண்ணே.

If in the above mentioned urine a foul smelling discharge is mixed up, it indicates inflammatory condition of the female and male genitalia or a stone in the bladder either already formed or forming.

விந்துவைப் போன்றநீர் விறல்கப நோயையும்
பந்தித்த சந்நி பாதத்தையுந் தரும்.

If the urine is like semen, it denotes *Kapha* diseases or *Sannipatha*.

விடுமுழி உவருருவிடின் அது கொடிதே.

If the urine does not leave a deposit on drying, it is of pathological import.

தக்கிரம்பால் எனத் தனித்தனி யொழுகுமேல்
உக்கிர வெப்பத்து உதிகால நீரே.

If the urine resembles either butter-milk or milk, it indicates excessive heat in the system amounting to grave results.

தீப்புலால் சழுநீர்ச் செயல்எனிற் குண்டிக்
காய்த்தூர்ப் பலத்தால் கதித்தநீராம் அத்
தூர்ப்பலம் கபமுஞ் சோரியுங் கொதிப்புறப்
பற்பக லாகப் பையப் பதிந்ததே

If the urine resembles the washings of putrid mutton, it denotes renal insufficiency; it is due to vitiation of *Kapha* and *Raktham.*

தடிக்கினு நெய்யின் தன்மையொத் திடினும்
படிக்குள் இரண்டு பாணியுந் தீதே.

If the urine is thick and viscid like ghee, it is incompatible with life.

Other Schools of Thought :— (மதாந்தரம்)

பாண்டரி நெய்யரி பற்றிய நீரெலாம்
தாண்டரி நோயே தருமெனுஞ் சிலநூல்.

If the urine is pale with a shadow of red, it indicates *Vatha* diseases.

கனிபொம்மன் மாசுக் கனிநிறம் போன்றும்
கனத்த சௌவீரக் களையது போன்றும்
சனித்த எண்ணெயின் தன்மையைக் போன்றும்
சாரியநீர் இவை சீரியமெய்க் குளாம்
பித்தப் பிணியாற் பிறந்தன வென்றே
சித்த சாரணவம் தெளிய விரிக்குமே.

If the urine resembles the colour of *Bombali-masu* fruit (*Citrus decumana*), has a shining like calomal and has an oily consistency, then it indicates the presence of *Pitta* diseases for a long time in the body as stated in *Siddha-Saranavam.*

மந்திரி நிறமாய் வரப்படு நீரும்
உந்திய எருவையை ஒத்துகு நீரும்
பித்தகா மாலைகளால் பிறந்தன வெனச்
சித்த வித்தியா தரம் செப்புமே.

If the urine is yellow and also red as blood, it indicates jaundice of *Pitta type* as said in *Siddha-Saranavam.*

அருச்சுனத் தொடுநுரை புற்புதம் எழினும்
அருந்துநீ ரொக்கினும் அவியினை ஒக்கினும்
கலப்பெழில் உறினும்அக் காண்டம் கபநோய்
தலைப்படும் காலைச் சாரும் என்பரே.

If the urine is pale with froth and bubbles or clear like drinking water lacking salt or like oil or with a. play of colours, it indicates the origin of kapha diseases.

இன்னும் அம்மதமே இருந்தொந்த நீழல்நீர்
தன்னில் கருமைச் சாயை உண்டென்னுமே.

If in the above mentioned colour there is a shade of black, then it denotes *Dwanda* diseases.

புண்ணீர் மேகப் புன்கண்மாப் பிணி
நண்ணில் நித்திய நாதீயம் ஆமெனும்.

If the urine contains blood, then it indicates *Rakthameham* or *Meharogam* as stated in *Nithyanadeeyam*.

சுரப்பிணி அரிசனம் தோயங் காட்டுமே.

In fever urine will be yellow.

சுரமேனும் மேகத் துனியேனும் இருக்கில்
பெருகிய மூத்திரம் பிரிக்கும் உயிரையே.

If patients suffering from high fever or *Meharogam* were to pass large quantity of urine it is of grave prognosis.

Relative Density :— (நிறை இலக்கணம்)

அற்பமும் கனமற் றதிதெளி வுறுமெனின்
வற்புறு சீதளம் மன்னிக் கனத்துக்
கபத்தை இளக்கலால் கண்டநீர் இஃதே.

If the urine is neither too thick nor too thin but very clear, it is due to vitiation of *Kapha*.

இதுநீர்ப்பை இந்திரியவுழி இவற்றுண்
முதிர்துர்ப் பலத்தான் முளைத்தெனக் கொள்க.

The above mentioned urine also indicates the weakness of bladder and seminal vesicles.

அன்றியும்,
மிக்குத் தடித்து விழுமேல் மலங்களின்
நற்குண வழுவை நன்கோது நீரே
இதனால்,
வீக்கமும் பிடகமும் மேவிடும் இவைமறு
நாட்குநாள் அல்கின் நலனாம் இவரின்
தொடில்சுடுங் கொடிய விடச்சுரம் அண்ணுமே.

If the urine is thick it denotes the vitiation of all the three *Doshas*. This will lead to inflammation or boils. If it decreases day by day the prognosis is good, if not it will lead to pyrexia or hyper pyrexia, (Visha Jwaram).

மிகத்தடிப்பும் மிகத் தேறலும் இன்றெனில்
சுகத்தைத் தருமெய்ச் சுபாவநீர் நன்றே.

If the urine is neither too heavy nor too light and neither too thick nor too thin, it is considered to be normal:

Odour :— (மண இலக்கணம்)

ஓதமணத் தொடவ் வோதம் ஒத்திறங்கும்
சீதளங் கம்பிய தேகி களுக்கே.

If the urine is having an odour as emenating during a river in floods and also is passed in large quantities, it denotes an increase of *Kapha*.

வெய்ய துர்க்கந்தம் வீசுநீர் மூத்திரப்
பைநாளம் இவற்றைப் பற்றுபுண் குறியே
அம்மொழி யின்றெனி னனிலமே முதலிய
மும்மலச் சுதமே மூலமென்று ணரே.

If the urine has a foul odour, it denotes inflammation of urinary passages or the vitiation of all the three *Doshas*.

மட்டிற் புளிமண மணந்திழிந் திடினல்
துட்டினப் பிணிபிணிப் புற்றெழு நீரே
முடிவிலவ் வுட்டின முளரிவா யறல்பொற்
படு சீதளச் சம்பந்த மாக்கிடுமே.

If the urine has an odour like tamarind, it indicates excess of heat. This in the end will lead on to the mingling of coldness or *Seethalam*.

இனித்த மணத்தோடிறங்கி னெய் மிக்குச்
சனித்த விருத்தியைத் தழுவிய நீரே
அல்லதவ் விருத்தியாலாம் பிணிக்குழாத்த
தல்லதில் லென்ப தாய்ந்தோர் மதமே.

If the urine has a sweetish odour, it indicates the vitiation of *Raktham* or a group of diseases due to excess of *Raktham*.

கருமான் மணமே கபமுமென் றுலது
குரவன் சுபாவ குணங்கெடு நீரே.

If the urine has an odour that comes from the black deer, it indicates the vitiation of *Pittam*.

புலால்மணங் கமழிற் பூதியப் பகைக்கே
டலாதிலை யந்நீரக் குணமின் றெனில்
அடையுந் ததியென் றறையத் தான்மிகக்
கெடுதி யெனவுங் கிளக்குஞ் சுருதியே.

If the urine has an odour of flesh it denotes the destruction of lipoidal tissues of the body. If it is not so then it will bring about that condition very soon and it is of bad prognosis.

Froth :— நுரை (இலக்கணம்)

பந்தம் மெய்ப்பசை இளகப்படும் பருவத்
தந்தர்ப் பூதமாய் அநிலம் மூத்திரத்தில்
சம்பந்தப் படும்தகி நுரைப் புனலே.

If the urine has much froth, it denotes the destruction of lipoidal tissues of the body and also that *Vatham* has become vitiated.

அந்நுரை பீதம் கருமை செம்மை
மன்னுறு நிறந்தரின் வரும் காமாலையென்
றுள்ளிப் புரப்பர் ஒப்பில் பிடகரே.

If the urine having froth is yellow, black or red, it indicates the impending origin of jaundice.

நுரைதேய்ந் தொழுகுநீர் நுவலரும் மும்மலம்
கரைய இளகிடும் காலத் தென்னே.

If the urine has little or no froth, it denotes the vitiation of all the three doshas.

Quantity ;—(எஞ்சல் இலக்கணம்)

இயற்கைநீர் சுருங்கினும் இதுவும் சலப்பொருள்
செயற்கையாய் அருந்தினும் சிறுத்தநீர் இதுவும்
பாண்டுநோய்ச் சம்பவத்தைத் தரும் இதில்
தூண்டுறுப் பேதியும் சோர்வும் பிறக்குமே.

If the urine is diminished in quantity without any obvious reason or if it continues to diminish in spite of drinking large quantity of fluids, then it indicates anemia (*Pandu*). This may lead on to intractable diarrhoea and langour.

OIL DROP TEST. (நெய்க்குறி)

நிறக்குறிக்கு உரைத்த நிருமாண நீரில்
சிறக்க எண்ணெய் ஓர் சிறுதுளி நடுவிடுத்து
என்றுறத் திறந்தொலி யேகாது அமைத்ததில்
நின்ற திவலைபோ நெறிவிழி அறிவுஞ்
சென்றது புகலுஞ் செய்தியை யுணரே.

With the same rituals the urine is collected and it is placed in the direction of sun rays where there is no draught of breeze. A drop of oil is let at the centre of the surface and is followed.

அரவென நீண்டிடின் அஃதே வாதம்.

If the oil drop lengthens like a serpant, it denotes the vitiation of *Vatha.*

ஆழிபோற் பரவின் அஃதே பித்தம்.

If the oil drop spreads like a ring, it denotes a disorder of *Pitta.*

முத்தொத்து நிற்கின் மொழிவதென் கபமே.

If the drop stays like a pearl, then it denotes a disorder of *Kapha.*

அரவில் ஆழியும் ஆழியில் அரவும்
அரவில் முத்தும் ஆழியில் முத்தும்
தோற்றில் தொந்தத் தோடங்க ளாமே.

If the pattern is like a snake in a ring, or ring in a snake, pearl in a snake or pearl in a ring, then it is due to Dwandam.

அழுந்து நெய்த்துளி அதுவுழும் மலத்தில்
எழுந்த குறிக ளெல்லாம் ஒன்றில்
தோற்றுவதும் முத்தோடம் என் றுன்னே.

If the oil drop sinks without spreading, it denotes *Thridosham.*

GOOD PROGNOSIS

விருத்தப் படிவமும் தரித்துப் பரவலும்
தெரித்த நெய்க்குறிக்கு இனிவருத்தமென் உலகீர்
ஆங்கப் பரவல்போல் நீங்கும் எப்பிணியுமே.

If the drop spreads circularly and slowly, then the prognosis is good but the patient will recover from the illness only slowly.

அரிவாகு நத்தை அரியா தனத்தை
அறிதரு குறியால் மறுவின்று உயிர்க்கே.

If the drop assumes a pattern resembling Vishnu's Conch or his throne, then the prognosis is good.

நாணயச் சத்தியை வீணையைக் காணினே
நாகுயிருடல் விடுத்து ஏகினும் மீளுமே.

If the oil drop assumes a pattern resembling umbrella or veena then the prognosis is good.

முல்லை யரும்பு முளரிப் பூவுஞ்
சொல்லிய துளியுள் தோற்றிடு மாயின்
இல்லை யில்லைநோ யென்பது சரதமே.

If the oil drop assumes a pattern resembling jasmine flower or lotus, the prognosis is good.

BAD PROGNOSIS.

எண்ணெய்விடின் சரேல்எனப் பொங்கிப் பரவின்
திண்ணம் அப்பிணிக்குச் சிகிச்சைஇங்கு இன்றே.

If the drop spreads very rapidly and foams, the prognosis is grave.

விடுதுளி சிதறி வெவ்வே றொன்றுமல்
கடுகெனப் பரவின் கைவிடல் முறையே.

If the drop scatters itself into small particles immediately, it is of very grave prognosis.

அவியும் முத்திரமும் அணைந்தொன்றின் ஆவி
அவியும் என்றல் கௌதமர் அறையே.

According to Gauthama if the drop mixes well with the urine, then it is of grave prognosis.

ஓதும் நெய்த்துளிஉள் அழுந்தில் தீதென
மாதவ கற்பம் மனனப் பயிலுமே.

If the oil drop sinks in the urine according to Madhavakarpam it is of bad prognosis.

அம்புகட்கம் முசலம் சூலமொடு வாள்
கும்பம் வெள்ளிலையென குறிபடின் தீதே.

If the oil drop assumes a pattern like arrow, sword, iron-rod (pounder) trident, dagger, pot or betel leaf, it is of bad prognosis.

எருதறு கியாளம் இபம்புலி மறிஅரி
நரன் அரவு அழுங்குபுள் நரிமர்க்கடம்கரம்
பூசைதேள் இவற்றைப்போல் உரு தோற்றின்
பேசுவ தென்னைப் பிணிதணி யாவே.

If the oil drop has a pattern resembling bull, lion, dinasaurs, elephant, tiger, goat, pig, man, snake, tortoise, bird, fox, monkey, donkey, cat, or scorpion, then it is grave prognosis.

PROGNOSIS GOOD OR BAD.

சல்லடைக் கண்போல் தனித்தனித் துவாரமாய்ச்
சொல்லுமுன் அற்றிடில் தொடல்என் கரம்எனக்
கௌதமர் உரைப்பினும் கபத்தால் கண்படும்
தவியா அணங்கது சாத்தியம் என்பரே.

If the drop splits into number of particles like a sieve even on touching the surface, *Gauthama* says that there is no use treating the patient; since it is due to *Kapha* the patient will recover. This is another view.

சாற்றிய கபத்தினுக்குச் சல்லடைக் கண்போற்காணும்
தோற்றிய திரிதோஷந்தான் சொல்மூன்று குணமுங்காட்டும்
வேற்றொரு துளியாய் நின்றால்விருதாகச் சாத்தியந்தான்
ஆற்றியேமெள்ளப் பார்த்தால் அதுசுகச் சாத்தியந்தான்.

In diseases of *kapha* origin the oil drop will assume a pattern like a *sieve*; in cases of *Thridosha* it will show all the three types; if it divides into minute particles the patient will be cured with some difficulty.

நீரினிலமிழ்ந்து போனால் நிகழ்ந்திடு மசாத்தியந்தான்.

If the drop sinks in the urine, it is of grave prognosis.

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Preliminary work with Cine-Photography in the Research of the investigation of urine :—

Soon after obtaining the sanction of the Academy to work on the problem of the examination of urine to assess the diagnosis and prognosis of diseases, we, in consultation with the directors, chalked out a permanent scheme of work in the shape of a Tabular Statement incorporating into it, as far as practicable, the necessary methods both according to Indian and Western systems that should be followed as a routine measure, in every sample of urine taken up for investigation. Thereupon, we requested the Academy to supply us a register in which the above mentioned tabular statement was printed and we got one such.

The next immediate problem that confronted us was the nature of photographs of the behaviour of a drop of oil on the surface of urine. For this we took a spool of 50 feet of Super X panchromatic Cine-Kodak film following a drop of oil in two different samples of urine. This was simply to know how far the spreading of oil could be photographed.

On the 27th of August 1941, after offering puja to the Goddess of Learning, Sri Sarasvathi, we started work in the Clinical Laboratory of the Government Indian Medical School Hospital. Drs. C. N. Kuppuswami and C. Dwarakanath helped us to start this work. Our exposed films were processed by Messrs. Kodak & Co., Bombay, and we projected the films in the Kodascope. We found that it was possible to photograph the oil drop on the surface of urine and follow up its reactions. It was observed that in the first film our technique was not quite satisfactory, since the distance between the eye of the Camera and the oil happened to be a bit greater than was desired. Hence we took another set of photographs with another spool of Super X panchromatic 8mm. Kodak film. This time we concentrated our attention on the following facts :—

(1) The dish containing urine be kept in bright sunlight and the drop of oil be allowed to fall gently on it.

(2) The Camara be focussed to the drop of oil so as to keep the drop in the centre of the field to follow its behaviour on the surface of urine.

(3) The spread of the drop of oil was followed until it was no longer seen.

50 feet of film were exposed for a drop of oil in 3 different samples of urine and the film was sent for processing to Bombay. When the film was projected it was found that the following three difficulties cropped up in photography;

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(i) The Camera did not seem to have taken pictures nicely at a close quarter of 1 foot.

(ii) At a definite stage the distinction between the oil drop and urine was not made out easily.

(iii) Error of parallax.

Thereupon we told the joint secretary about this and he communicated the matter to Messrs. Kodak & Co., Bombay for technical advice. A few days later the local representative of Messrs. Kodak & Co., met us and gave the following suggestions :

(a) For this kind of photography, a Kodak Magazine Camera would have to be used with a sharper and more powerful lens,

पुस्तकालय गुरुकुल कांगड़ी

(*b*) Colour films would give better results.

(*c*) The Camera should be fixed and the distance between the eye of the Camera and the drop of oil should be well adjusted and that distance should be kept constant in our subsequent exposures.

Hence, before we could start the regular work, we started to get over these difficulties. We have arranged with the Technician of Messrs. Madras Photo Stores, our film suppliers, to come on Thursday, the 23rd instant, when we could be taking a colour film to note the behaviour of a drop of oil on the surface of urine. We obtained one spool of colour film and two of ordinary films to take photos once again.

The problem of differentiating the oil drop from the colour of urine at a particular stage of experiment is still to be solved. We are at present using a Cine-Kodak with 3·5 lens (Camera) and a Kodascope belonging to Government Indian Medical School Hospital. As already mentioned the technical advice on this matter is for a Kodak Magazine Camera to get 'close-up' pictures.

The approximate cost of entire Research will be as follows :—

	Materials.		Rs.	A.	P
I.	Kodak-Magazine Camera	...	400	0	0
II.	Kodascope (sharp projector)	...	225	0	0
III.	Films, 3 ordy, and 3 colour films per month	...	720	0	0
IV.	Editing and printing of titles and short descriptions relevant to research	...	100	0	0
V.	Still photographic reprints of some typical specimens	...	40	0	0
VI.	Procelain or enamel dishes	...	50	0	0
VII.	Incidental expenses	...	115	0	0
			1,600	0	0

This approximate sum towards the cost of research will have to be provided.

C. B. HANUMANTHA RAO.
Y. KONDAL RAO.

Liberty Press, Madras,

Issue No. I

TRANSACTIONS

of

The Academy of Indian Medicine

MADRAS

(1941—'42)

पुस्तकालय
गुरुकुल कांगड़ी

RAJAYAKSHMA

(Pulmonary Tuberculosis)

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By

C. DWARAKANATH, L.I.M. (Madras) Z. T.
(HAMBURG 'VARSITY)

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RAJAYAKSHMA OR PULMONARY TUBERCULOSIS

The disease known in our country as Rajayakshma, Kshaya, *Urakshatha* or Sosha and in the Western world as Tuberculosis has always been a very interesting and an absorbing problem to all physicians ancient and modern. It has presented itself as a very complex phenomenon. The large mass of investigations carried on with regard to this disease in the West during the last 150 years have yielded a fund of information regarding the several aspects of the disease, though in themselves quite inconclusive. There have been different schools of thought as to the why and wherefore of this disease in modern times. Compared with the maze of facts clinical, pathological, and experimental of the present, the ancient version of the disease seems to be comparatively conclusive. It will therefore be of interest to examine its etiology and treatment from the Indian and Western stand points of view.

History and the evolution of modern ideas of the disease :

The disease was known to have existed and understood in ancient Greece as is evidenced by earliest writings of the Greeks.[1] References to the havoc played by it are to be found in the Cuneiform tablets among the remains of Babylon. Long before the Christian era it was found by Egyptains. This is evidenced from the skeletons of the Egyptian mummies examined by Dr. Derry (3000 to 2000 B. C.) and Elliot Smith (1000 B. C.).[2] The germ responsible for this disease 'Tubercle Bacillus' was demonstrated in the tissues of the Egyptian mummies from the Herst collections at Cairo belonging to the 20th dynasty or so.[3] Biblical allusions as early as 1500 B. C. are today understood to refer to this disease. Hippocrates (460 B. C.) describes it as a febrile disease occurring mostly between the ages of eighteen and thirty-six, and as due to some constitutional defect which he called 'phthisical diathesis'. His contemporary Socrates, and Aristotle thought that it was a communicable disease. Aretaeus (250 B. C.) believed along with other ancient European physicians that the disease was associated with the emphysema and prescribed sea air and sea voyages for its cure. Celsus, 300 years afterwards, recommended a diet of milk, a change of air, and sea voyages from Italy to Egypt. Galen (A. D. 130 to 201) advocated fresh goat's milk with salt or honey and prescribed a stay in a warm climate and in the highland of Phrygia. During the Mohammedan era Rhazes described the several varieties of the disease. Avicenna and Serapion elaborated Galen's conception of the disease and its treatment and laid stress on the value of ass's and goat's milk and also prescribed syrup of poppies to relieve cough. After the Arabian period one passes over many centuries during which time medieval medicine in Europe remained more or less dormant and static. During the Renaissance, in Italy, France and Spain the infectious character of the disease was stressed and very stringent laws were brought into force against the spread of the disease and its victims. The extreme views of the contagious character of phthisis prevailed up to the 18th century and died down in the 19th. To the credit of the Englishmen it must be said that during this period the theory of contagion found no support in England. During the 17th century Richard Morton (1689), author of the reputed Phthisiologia, attributed consumption to some derangement in the blood.[4] Sibbald describing the incidence of the disease among the Scotch peasants in the 17th century attributed the disease to bad food, strong drink and excessive toil. Early 19th century physicians considered diathesis and morbid constitution as predisposing to tuberculosis. Dr. Benjamin Rush (1808) and Sir Thomas Watson[5] (1836) could not subscribe to the theory of contagion. *Hughes Bennett[6] the physician who introduced the cod liver oil therapy argued strongly that tubrcle was the result of the errors of nutrition, while Walshe thought it was due to the failure of digestion and chyle formation.* In 1883 the British Medical Association sent out to its members a questionnaire as to whether the disease was contagious. Out of 1,178 replies, 778 were given in the negative, and only 261 were in the affirmative, while 39 were doubtful. Laennec, the inventor of the stethoscope concluded from his own experience that the disease was not usually contagious. In the continent[7] Rokitansky, from a vast experience formulated the law of the phthisical habitus—a small heart in a long, narrow chest, dyscrasia of the blood favouring pleuritic and other exudates, a delicate structure of the arterial walls and of the skin. Then came Virchow, who founded the modern cellular pathology in direct antogonism to humoral pathology and blood dyscrasia of the ancients. Villemin in France, Cohnheim and Klebs in Germany—made experiments on rabbits and guinea-pigs to prove the communicability of tuberculosis from one animal to another, and threw out suggestions of a virus from without as the cause of tubercle. While the professional views were swaying between constitution and contagion, Robert Koch in March 1882, announced at the Berlin Physiological Society his discovery of a bacillus in human and bovine tubercle, which seemed to confirm the views of infection held before in doubt. Under the spell of the brilliant work of Koch and the French school, various remedies to destroy infection were set on foot all over the civilized world and bacteriology opened up a vast field of experimental research to prove the microbic origin of the disease.

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[1] Tuberculosis Internationale, June 1914.

[2] G. E. Smith and M. A. Ruffer, 1910, 'Pott'sche Krankheit an einer Agyptischen Mumie', ZUR HISTORISCHEN BIOLOGIE DER KRANKHEITSERRIGER.

[3] Medical contributions to the Study of Evolution, by T. G. Adami.

This brings us to a very hotly debated question (1) whether the disease is entirely due to the parasite as described by Robert Koch or it is merely one of those incidental factors very closely associated with the disease; or (2) whether the soil or the human body is primarily responsible for supplying an opportunity for these incidental factors to complicate the issue and create a specificity to the disease. It will therefore be worthwhile for us to have an idea of the parasite in question.

The tubercle bacillus is described as *a parasite in the full sense of the word, living and thriving only in the bodies of animals and men and perishing outside the animal body.*[8] Maurice Fishberg says that it has not been decided to which group of micro-organisms it belongs and in fact they do not as yet have a classification of bacteria which is completely satisfactory to all who are competent to judge. It may be said to belong to acid fast bacteria of which there are many varieties, may be classified with Trichomycetes, while some consider it an intermediary between the true bacteria and the lower fungi, hypomycetes. The morphological variations of the tubercle bacilli are correlated with their type and virulence, whether human, bovine or avian and the media in which they have been cultivated. In preparations made from sputum expectorated by tuberculous patients, the form appears as a slender rod mostly straight but sometimes curved, about $\frac{1}{4}$ to $\frac{1}{2}$ the diameter of a red blood corpuscle or 1/500 mm. in length on the average. These rods are mostly rounded on the two ends and occur singly, in pairs or heaps. They are non-motile and non-flagellated. They have an enveloping or capsular, fatty or waxy substance. The germ multiplies by elongation to transverse fission and not by spore formation. It is mostly stained by Ziehl-Neelsen method. Calmette and his followers were of opinion that chronic tuberculous disease characterised by pathognomonic pathological changes in tissues especially giant cells and showing the presence of acid fast bacilli in the various organs, is by the end stage of an infection caused by the ultra-virus which is at first invisible in the microscope and which gradually changes into non-acid fast dustlike granules and finally evolved into the pathognomonic acid fast tubercle bacilli.

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[4] Contributions to the Physiological Theory of Tuberculosis, by Charles Creighton, p. 2.

[5] The Causes of Tuberculosis, by Louis Cobbett, p. 50.

[6] Charles Creighton—Contributions to the Physiological Theory of Tuberculosis

[7] Tuberculosis Internationale, June 1914.

[8] Pulm. Tubercle, Vol. I, page 58, by M. Fishberg.

The life cycle of the Germ :

[9] Sweany and Khan determined the pleomorphic nature of the bacilli. They found that a variety of forms may be described which appear to be stages in a complicated life cycle and that *the environment in which the organism finds itself has a profound influence on its appearance and its acid fastness.*[10] The main results of these researches have been to relegate the tubercle bacilli of Koch's classic description to that of a comparatively minor place in the pathogenesis of tuberculosis . . . It seems likely that the chromophobic (unstable or mostly unstable and also therefore non-acid fast) tubercle bacilli are of the greatest importance in the development of tuberculosis, experimental and clinical. It has been found that light, especially sunlight decomposes the fatty substance in the bacilli and destroys them altogether. On the whole the bacilli may retain their vitality for a considerable time, if not in exceptionally *unfavourable surroundings.* In the latter case the growth is soon hampered. Four different varieties of this organism have been identified, *viz.,* human, bovine, avian and reptilian. Of these the human type of the bacilli is found in vast majority of forms of tuberculosis in human beings; in adults, pulmonary tuberculosis is almost by this virus.

Other varieties of similar acid fast bacilli have been identified and are called by the name pseudo-tubercle bacilli. They are (1) Smegma bacillus and is found in the secretions of the external genitals, mammae, etc. (2) Hansen bacillus, which is said to cause leprosy; and (3) Moller's grass bacilli otherwise known as Timothy grass bacilli. Inoculations with this type of bacilli produces lesions exquisitely resembling tubercles. All these bacilli have been found under favourable environmental conditions to cause lesions which resemble real tubercles in all respects with the difference that they do not spread beyond the area of inoculation. Another difference which has been described so that while genuine tubercles are of a proliferative character, these pseudo-tubercles are of a more exudative and inflammatory character having a tendency to abscess formation. Therefore it has been found to be very difficult to differentiate the pathogenic from the non-pathogenic acid fast bacilli microscopically; even the experienced often cannot tell the difference when examining a smear on a slide which shows that clinically when the non-pathogenic bacilli are found they are apt to lead to erroneous diagnoses.

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[9] **Am. Rev. Tuber 1931, 24, 73 and 1928, 17, 53.**

[10] **Fishberg Pul. tuber, Vol. I. p. 65.**

Four different modes of infection have been suggested; *i.e.*, (1) contact infection or inoculation into the skin or mucous membranes; (2) inhalation through the respiratory passages or aerogenous infection; (3) infection through the digestive tract, intestinal infection; (4) Germinative, or placental infection, the bacilli being derived from the parents before the birth of the individual. Talking of the first variety, it has been shown by great workers like Lewandowsky that the skin is not a culture medium for the tubercle bacillus, it does not permit it to grow and proliferate. Available evidence from great workers tends to show that infection through the skin is hardly of any importance in the etiology of tuberculosis of visceral organs, notably the lungs.

Infection by inhalation:

Though many of the ancient authorities on medicine in Europe believed that the disease was communicable through air or inhalation it was only the animal experiments of Koch and Cornet[11] which were able to prove that dust containing tubercle bacilli derived from desiccated sputum is highly infectious to guinea pigs. On the basis of such experiments rested the entire inhalation hypothesis of tuberculous infection. The fact that diffuse daylight, especially sunrays, kills tubercle bacilli and soon renders them avirulent, would largely exclude infection through sputum deposited in the street and in large sunny rooms. Many valid objections have been raised against the theory that desiccated tuberculous sputum is the main source of infection in man. Flugge[12] and many others have shown that in the ordinary course of human events things are not so simple as stated by Cornet and Koch. Such large quantities of sputum as were used by Cornet in his experiments on guinea-pigs are exceedingly rare, if ever found in the most squalid of dwellings. Investigations done by Flugge, Neisser, Kohlisch, Lange, Neufeld[13] and others have not yielded the same results as those reported by Koch, Cornet and their followers. It was found that in houses inhabited by consumptives, a sputum deposited on the floors is not often perfectly dried and thinly pulverized, capable of rising with the dust to the height of 5 or more feet from the ground. Moreover conditions in insanitary homes are as a rule not conducive to drying of the sputum soon after it has been eliminated by the consumptive. And if it takes time to dry, it must be remembered that the bacilli loose their virulence within ten days, owing to putrefactive processes on the floors of filthy houses and the diffuse light which acts during the day or artificially during the night. It is also worth mentioning in this connection that in an average house there are no air currents strong enough to raise the dust to the height of about 5 feet and more. It is a very incredible fact that it is exceedingly rare to find a house where proper precautions are taken as to expectoration in which the collected dust shows virulent tubercle bacilli.[14] Even the houses inhabited or frequented by consumptives—sanatoriums, dispensaries, railroad stations, factories, cars, etc.—no virulent tubercle bacilli have been found in the collected dust in most cases investigated. Guinea-pigs, which are very susceptible, were inoculated with dust collected in houses inhabited by consumptives and it was concluded from such experiments that it has no significance in the origin of tuberculous disease. Wagner collected dust in a sanatorium at Zurich, in such places in which the air stream could have dispersed it, and injected it intraperitoneally into guinea-pigs and found that in only 3·5 per cent of cases did the infection take place. The experiments of Brown, Petroff and Pasquera in which the dust collected by a vacuum cleaner from a large rug in the living room of the Trudeau Sanatorium proved negative when injected into a guinea-pig. Identical results were obtained with the dust from a room in the infirmary occupied by a patient with numerous tubercle bacilli in the sputum and a cough so explosive that the mouth was rarely covered. Collections of dust before the daily cleaning by swabbing with sterile swabs, the bed, tables, chairs, bed frame, corners of the rooms and walls near the patient, were washed in sterile broth, the washings treated with normal sodium hydroxide, incubated for one half-hour, then neutralized with normal hydrochloric acid, centrifugalized, and the sediment divided into three portions. One was inoculated into Petroff's gentian violet egg medium, another stained on a slide for microscopic examination, and the third inoculated subcutaneously into the inguinal region of guinea-pigs, two for each swab. In all 24 animals were used. All the slides were negative as regards tubercle bacilli and none of the 24 animals showed any signs of tuberculosis when carefully examined at post-mortem. These experimental facts exclusively prove that infection by inhalation is rare in spontaneous tuberculosis in human beings.

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[11] Verhandl Berl. med. Gesellsch., 1899, 30, 91.

[12] Ztschr u. Hyg. w Infections Krankh, 1909, 30, 107.

[13] See Neufeld, Origin and Dissemination of Tuberculosis, Harvey Lectures of 1926-1927, p. 27.

Droplet infection :

The moist droplets eliminated by consumptives while speaking and especially while coughing and sneezing, may be inhaled by persons who happen to be in their proximity. Flugge[15] and his followers, who have done considerable experimental work on these lines, are satisfied that under natural conditions the dissemination of tuberculosis from man to man, droplet infection is the most common mode. But Maurice Fishberg holds that the practical bearings of the problem of droplet *vs.* dust infection in the most recent investigations, have by no means clarified the issue.[16] Careful investigations have shown that the air exhaled by consumptives during ordinary and quiet breathing is free from tubercle bacilli, but the moist droplets eliminated from the mouth while talking, coughing, sneezing etc. do often contain the germs which may remain floating in the air for some time. He also says that the problems of droplet infection are many and it may be said that no agreement has yet been arrived at by all who are entitled to an opinion. Experimentally, it has been shown that when a tuberculous patient coughs at a cover glass, bacilli are found on the slide in a variable proportion of cases. For this purpose, the cover glass must be held close to the mouth of the patient. Some found that while when held 40 to 80 cm. away from the patient's mouth 30 per cent of the cover glasses showed tubercle bacilli microscopically. No bacilli are deposited when it is held one metre or more away from the mouth of the patient. Engelmann showed that when the slide is held at the side of the patient's face, negative results were obtained even when the distance was but 30 cm. from the patient's face. In America J. B. Rogers obtained positive results at a distance of 15 inches, but Brown, Petroff and Pasquera met with failure, while making somewhat similar experiments.

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[14] **M. Fishberg, Pul. Tuber, Vol. I, p. 97.**

[15] **Die Verbreitungsweise and Bekamfung Der Tuberkulose auf. Grund experimenteller Untersuchungen, Leipzig, 1908.**

[16] **M. Fishberg. Pul. Tuber, Vol. I. p. 98,**

Another method is by placing the susceptible animals before consumptives. Flugge immobilized guinea-pigs and made the tuberculous patients cough directly into their mouths. The animals became tuberbulous in six out of 25 cases. Moller had tuberculous patients cough at guinea-pigs daily for several weeks and in two out of 14 cases was tuberculosis thus transmitted and it is therefore argued that these experiments are open to question. The animals were held tightly for hours directly exposed to the faces of the consumptives who coughed directly into their open mouths. Such exposure never occurs in human beings, except perhaps in cases of tuberculous mothers holding their crying babies on their arms, coughing directly into their open mouths, as may be observed now and then among certain classes, but after all this cannot be considered very common. It is therefore obvious that only when contact with the consumptive is very close, intimate and prolonged, which in ordinary life occurs as a rule, only in mothers with suckling infants, or between husband and wife, droplet infection may become a serious menace and even in these cases there are natural safeguards.

Considering all these available evidence at their face value, it appears that inhalation of dust containing tuberculous sputum, or of droplets expelled by consumptives while talking, coughing and sneezing, may infect a healthy person, yet the evidence that these are the most frequent modes of dissemination of tuberculosis is inadequate. It must also be borne in mind that in spite of the various dishormonies which may be found in the structure and functions of the human body, the respiratory tract is provided with a most wonderful protective apparatus for the prevention of the entry and implantation of the bacilli in the deep respiratory passages. As a matter of fact, no organ in the body except the central nervous system is equipped with better safeguards in this respect. The investigations of normal lungs have shown that they are germ-free, the few bacteria which may remain within for any reason are, under normal conditions, well cared for by the extensive lymphatic apparatus which surrounds all the bronchi and blood vessels, even the terminal bronchioles, and takes up bacteria, destroying them or at least rendering them innocuous. The tubercle bacilli are rarely found in the lungs of animals compelled to inhale dust containing the germs. On mucous membranes they do not invariably cause disease is proved by another fact. These have been found on the mucous membranes of the nose, throat and mouth of healthy individuals. Many workers like Nobel W. Jones, Strauss, and Alexander found them in large numbers in normal people, particularly those suffering from ozena, but they had no symptoms or signs of tuberculosis. These facts taken in together with the fact that tuberculosis of mucous membranes of pharynx, nose and mouth is exceedingly rare even in consumptives, show that these structures possess a certain amount of natural resistance against the disease. A lymphatic apparatus of normal structure and function evidently insures against the implantation and pathogenic action of all kinds of bacteria in the respiratory passages. Otherwise all of us should have succumbed to various microbic diseases including tuberculosis. It is only when the natural protective forces fail that tuberculous disease may be caused in this manner. The reasons for the failure of experimental investigations to adequately solve the problems of the areogenous etiology of phthisis are evident when we bear in mind that pulmonary tuberculosis as met with in human beings, showing isolated foci which extend slowly downward in the lungs, never occurs spontaneously in animals, nor has it ever been induced artificially or experimentally, in animals.[17]

Hematogenous infection or blood-borne infection :

Many Western authorities look at tuberculosis as hematogenous in origin. The bacteria are assumed to enter the body from any point through the respiratory or alimentary tract or even through the skin and are carried by the blood stream until they reach a point, where the tissues have a low power of resistance, an organ which offers a favourable soil for the growth and action of these micro-organisms. From the unanimity of the frequency of lung tuberculosis, it is assumed that in great majority of people the lungs offer a good breeding point for the germs. The localisation of the bacilli is thus said to be accomplished in a like manner as their localization in joints, the peritoneum, the meninges, etc. by the blood stream. *Aufrecht, a renowned German worker, holds that the primary tuberculous lesion is always in the vascular walls, which are affected by bacilli brought to them by blood stream.*

Infection by ingestion :

By far the most important mode of hematogenous infection in tuberculosis is suggested to be by ingestion of tubercle bacilli, although it by no means excludes the air passages as portals of entry, because germs inhaled through the mouth, nose and throat may be swallowed and passed into the blood stream through the mucous membranes at any point of the gastro-intestinal tract. However, in the vast majority of cases, it would be with food. Simple as this theory looks, many valid objections have to be considered before accepting it. While the gastro-intestinal secretions may and usually interfere with their rapid proliferation and so may any fermentation in the intestinal tract, while the peristaltic movements of the intestines may seem to remove them from the body without necessarily killing them. Many experiments have been done to prove for and against this conception. Different authors suggested different parts of the alimenary canal from the mouth to the rectum, as portals of entry of the bacilli, which are taken up by the blood and carried to the lungs where they finally stay and cause the disease. Some have laid the blame at the doors of irritated gums during dentition of children as offering a good way of entry for the bacilli, others found the tonsils guilty, particularly the pharyngeal tonsils. From such and many other observations Maurice Fishberg concludes that the tuberculous infection including phthisis may be acquired by the ingestion of tubercle bacilli and that the digestive tract permits the passage of the bacilli which are carried by the blood and the lymph streams to the various points of least resistance, amongst which in the human being the pulmonary tissue appears to be the most vulnerable. However, he questions if this mode of infection is the commonest in spontaneous tuberculosis in human beings. We should not also overlook the fact established experimentally that large numbers of the germs are necessary to accomplish results ; the normal gastro-intestinal tract being

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17. M. Fishberg. Pul. Tuber, Vol. I, p. 101 to 102.

equipped to easily dispose of small doses of tubercle bacilli. Talking of milk infections, the danger thereof appears to be overestimated by many writers Two sets of facts prove this. There are countries in which the bulk of the population do not use cow's milk as a food at all, where pulmonary tuberculosis is not at all lacking. This is true in the case of Japan, China, India, Egypt etc. Secondly, when infection with bovine type takes place, the dangers thereof are slight even in children. According to Kossel,[18] during the period of 1905 to 1909, the Imperial Health Department in Germany investigatad 113 cases of tuberculosis of the udder in cows, it was found that in only 44 cases their milk was being consumed after being boiled. Altogether 360 persons consumed milk from these cows in a raw state (150 children, 200 adults and ten whose age was not ascertained). It was noteworthy, that of this, all but two remained healthy. The two infected were infants aged twenty-two and fifteen months. The parts involved in these infants were the lymphatic glands. General health of these children remained good. No symptoms of any general disease appeared when re examined one and a half and two and half years later, they were found to be thriving. Hess[19] in New York followed 18 children who drank milk in which tubercle bacilli were demonstrated and found that all but one remained free from tuberculous disease. This one child developed tuberculous adenitis. From all these data, it appears that the disease caused in children by the bovine bacilli manifests mostly in diseased glands, bones, joints, intestines and skin, while fatal pulmonary infections are extremely rare. Evidences in galore are marshalled to prove that the adult human is practically immune to the bovine type of tubercle bacilli even if his immunity to the human strain has not yet been established to the satisfaction of all. There are many authorities who are of opinion that mild bovine infection immunises the organism against infection with more virulent human type. Cobbet[20] points out that it is well known that immunisation with small doses of the virus occurs in diphtheria, 'the frequency with which tubercle bacilli are met with in England whether derived from sputum or milk is one of the means whereby the resistance of the people is raised above that of primitive peoples'. From the various data brought out consequent to enormous amount of experimental work done with the one object of shedding more and more light on the problems of tuberculous infection have not entirely or satisfactorily explained the mode of entry of the bacilli into the human body. The contradictions in the experimental results obtained by various investigators can be easily explained by the several methods pursued in investigations, the different cultures and infecting doses employed, the character of the culture media, the species of the animals used etc. Even after discounting all these factors, it remains a fact that hardly any of the experimental modes of infection have completely imitated natural conditions as observed among human beings.[21] Fishberg says "*that there is no agreement between the authorities as to the most common channel of entry of the tubercle bacilli*, and Romer, perhaps the most indefatigable in the field of tuberculosis, arrived at the conclusion that "*there is evidently some mode of transmission of this disease with which we are yet unacquainted,*" and work done since Romer has by no means clarified this subject—that in the human being the infection alone is not sufficient to produce disease, after all disease occurs only in a comparatively small proportion of persons infected with tubercle bacilli. These micro-organisms harm only those who are predisposed to the disease. *We are more and more becoming convinced that phthisiogenesis is more a problem of predisposition than of bacterial infection*".

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[18] Deutsch. Med. Wchnscher, 1910, 36, 349.

[19] Jour. Am. Med. Assn. 1911, 56, 1322.

[20] Tubercle, 1925, 6, 584.

[21] M. Fishberg. Pul. Tuber. Vol. I. pp. 111—112.

Just a few words about the pathology and clinical aspects of the disease as understood by the votaries of Western medicine. In post-mortem made on people who died of this disease, the involved structures showed definite changes involving variable quantities of small and hard nodules. These were named tubercles. The immature tubercles are found to be grey and translucent, the older ones yellowish, white and opaque. The microscopic section of a single tubercle presented characteristic structure of a fairly well circumscribed aggregation of cells. It is avascular. The tubercle is more or less made by epithelioid cells and there is a paucity of intercellular substance. The peculiarity of tubercle is the presence of multinucleated giant cells. The origin of this structure has been a matter of much debate for a long time. Under diseased conditions these tubercles undergo degenerative changes of the type of necrosis. The first stage of such a change is known as caseation, that is becoming cheesy. This is considered to be due to certain poisonous substances elaborated by the tubercle bacillus. When large areas of the involved organ caseates variable mass of the tissue substance separates and leaves potential cavities. The other change of which tubercles are said to undergo is calcification or getting stony by the deposition of calcium. Such calcified foci usually harbour virulent tubercle bacilli. Another change which is also possible is fibrosis, otherwise known as healing by scar formation of the ulcerated tissues. The evolution of the tubercle and the two different kinds of changes namely, destruction of the organs involved on one hand and healing as evidenced by calcification and fibrosis on the other, are correlated to the clinical symptoms and complication usually observed. There are several divisions of the disease into various stages but what finds currency now in Mid-Europe is as follows :—(1) *Primary complex,* meaning the initial localization of the infection, (2) a secondary stage of generalization with the development of hypersensitiveness and often the formation of hematogenous metastases, (3) relatively high immunity and isolated organ tuberculosis. In other words, the *primary complex* is generally fulminent with the picture of acute sepsis or caseous lobar or broncho-pneumonia as the case may be. *The secondary stage* is evidenced by the exudative nature of the lesion and marked with milliary tuberculosis or run a malignant course or may become chronic. *The tertiary stage :* In the course of time those who do not succumb to the manifestations of the primary or secondary stages (and they constitute the vast majority of infected individuals) gradually enter the tertiary stage, which is characterised by isolated tuberculosis, of which the lung form is the commonest. This is also marked by productive reaction and the development of specific granulation tissue. The clinical varieties of lung tuberculosis can be divided under the following : (1) Exudative tuberculosis, (2) Cirrhotic or fibrotic or productive or proliferative type of tuberculosis. The former occurs as an acute condition and the latter as chronic. The complications and accidents which occur during the course of the disease are (1) Haemoptysis, generalised bacterimea, and spontaneous pneumothorax.

Dr. C. Muthu thinks that the disease is simply "an expression of man's failure to adapt himself to rapidly changing new environments, The progressive changes in the evolution of man, from pastoral and agricultural to commercial and industrial life, were marked by an increase of wealth and comfort on the one hand and an increase of responsibilities and cares on the other; as civilized man further extended his conquest and widened his sphere of commercial enterprise into other countries, great upheavals took place in his physical and moral environment for which he was not prepared. It is this failure to meet new conditions and adapt himself to successive changes that has brought about social and economic evils which have accumulated with each generation............"

"More than a generation has passed away since Koch made his epoch-making discovery. The measures taken against the contagion of tuberculosis have not produced such a reduction in its morbidity as his followers had expected. **We are now beginning to see that the foundations of tuberculosis lis deeper than the presence of tubercle bacilli, and spiritual factors have an important bearing in the causation and the cure of the disease............**"

"Life is one. Mankind is one. Microrganisms are the product of environment. Disease is interrelated in the defective or the altered state of the blood, hence diseased conditions are within us and not outside. We carry within us the potentialities for good or evil. Life is manifested by resistance. Behind microbes there lie ten thousand activities of the endocrine glands and metabolism, and behind these again lie the operations of the mind and the spirit which man can control."[22]

Now coming to the examination of the subject from the standpoint of view of Ayurveda, it appears from the classical descriptions of the disease and its causes that the clinical manifestations have changed considerably from what they were in the ancient days. But what is evidently common between the Ayurvedic version of the etiology of the disease and that of modern, both intrinsic and extrinsic, appears to have many things in common.

The disease is said to have first occurred in Chandra, the King of the Constellations, who transgressed the laws of nature. It is said that he had about 27 wives who were all daughters of Daksha and who took no care for preserving his body. It is also said that Chandra liked Rohini, one of his wives, and not all and therefore Daksha, his father-in-law, became wrathful. His anger took the form of his breath and came in an embodied form from his mouth. The allusion here seems to be due to his indifferent ways of life full of dissipation without paying any regard to the fundamental laws governing the maintenance of health. Be this as it may, what is important to us of the present day is the disturbance in the equilibrium of the three important factors, functional namely Vatha, (dynamic) piththa and Kapha which are static requiring the help of the dynamic Vatha for their functions.

पित्तं पङ्गुः कफः पङ्गु पङ्गवो मलधातवः ।
वायुना यत्र नीयन्ते तत्र गच्छन्ति मेघवत् ॥

Sarangadhara Poorva, Ch. V, 25.

By Saptadhathus I mean structural or organic factors. The equilibrium of these essential factors is said to be disturbed by (1) excess of correlation, (2) absence of correlation and injudicious correlation in respect of objects, of acts, of senses and of time.

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[22] Pulm. Tuber.—Dr. C. Muthu—p. 5 to 10.

त्रीण्यायतनानि अर्थानां कर्मणः कालस्य चातियोगाऽयोगमिथ्यायोगाः ॥

Charaka, Suthra: XI; 37.

कालार्थकर्मणां योगो हीनमिथ्यातिमात्रकाः ।
सम्यग्योगश्च विज्ञेयो रोगारोग्यैककारणम् ॥

Ashta. Hru. I; 27.

"The basic causes for all disturbances of health in general and Rajayakshma in particular is dhoshavaishamyam."

रोगस्तु दोषवैषम्यं दोषसाम्यमरोगता ॥

Ibid 28.

These factors are important in determining several varieties of diseases. In other words, the subject and his reactions to hyper, hypo and perverse states of his own physical, mental and sense activities and also his reaction to time factor likewise, brings about a disturbance and disharmony in the delicate equilibirium of the *dhoshic* and *dhathu* balance, thus preparing *the body soil* as an appropriate medium for diseases.

कार्ययोनिर्धातुवैषम्यं तस्य लक्षणं विकारागमः ।

Charaka—Vimana VIII; 14.

All the great authorities of Ayurveda have mentioned four important causes which disturb the body soil in such a way as to cause *urakshatha.*

"Exertions in which strength beyond the ordinary measure is put forth, suppression of the urgings of nature, waste (of semen, blood and other constituent elements of the body), and inharmonious or incompatible food and drink."

इह खलु चत्वारि शोषस्यायतनानि तद्यथा
साहसं सन्धारणं क्षयोविषमाशनमिति ॥

Charaka—Nidana VI; 3.

Susrutha speaking of this says "the process of any wasting diseases in the system, repression of any of the physical urges, over-fatiguing acts and irregular and inadequate or inappropriate diet are the causes, which by disturbing the bodily dhoshas spreads vitiated fluids all over the body, giving rise to the disease".

क्षयाद्वेगप्रतिघातादाघाताद्विषमाशनात् ॥
जायते कुपितैर्दोषैर्व्याप्तदेहस्य देहिनः ।
कफप्रधानैर्दोषैस्तु रुद्धेषु रसवर्त्मसु ॥

Susrutha—Uthara XLI-8, 9.

Again he says "that sexual excess, grief, old age, over-fatiguing acts, toils of journey, inflammation and ulceration in the chest (uras) are included by certain authorities as the exciting factors of the disease."

व्यवायशोकस्थाविर्यव्यायामाध्वोपवासतः ।
व्रणोरः क्षतपीडाभ्यां शोषानन्ये वदन्ति हि ॥

Susrutha—Uthara XLI ; 16.

Talking of wasting, Charaka says "that habitual use of dry foods and drinks, fasts, insufficiency of foods, excessive grief, suppression of vegas and sleep, habitual use of dry udvarthana (unguents) and indulgence in bath without rubbing oils on the body, congenital tendency to waste or waste that follows diseases and excessive wrath that makes a man lean. Diseases of spleen, consumption, asthma, abdominal tumours, piles and other abdominal diseases, and also those diseases that affect grahani assail the emaciated man."

सेवा रूक्षान्नपानानां लङ्घनं प्रमिताशनम् ।
क्रियातियोगः शोकश्च वेगनिद्रा विनिग्रहः ॥
रूक्षस्योद्वर्तनस्नानस्याभ्यासः प्रकृतिर्जराः ।
विकारानुशयः क्रोधः कुर्वन्ति कृशं नरम् ॥
....
प्लीह कास क्षयः श्वासो गुल्मार्शांस्युदराणि च ।

Charaka—Suthra XXI; 10, 11, 12 & 14.

Charaka says that "the several dhathus are developed by their respective measures of heat. Each coursing through its own srothas nourishes the rest. In consequence of the obstruction of the srothas and the vitiation (in quality and quantity) of the blood and other dhathus and also the loss of inherent heat (Agni) in each dhathu Rajayakshma is induced. At such a time the food which is cooked in the stomach by Jatharagni (Koshtagni) becomes converted for the most part into mala (impurities), very little of it is converted into anything substantial."

यथा स्वेनोष्मणां पाकं शरीरायान्ति धातवः ।
स्रोतसा च यथास्वेन धातु पुष्यति धातुना ॥
स्रोतसां संनिरोधाच्च रक्तादीनां च संक्षयात् ।
धातूष्मणां चापचयाद्राजयक्ष्मा प्रवर्तते ॥
तस्मिन्काले पचत्यग्निर्यदन्नं कोष्टमाश्रितम् ।
मलीभवति तत्प्रायः कल्पते किंचिदोजसे ॥

Charaka Chikitsa VIII; 36, 37, 38, 39.

रसः स्रोतःसु रुद्धेषु स्वस्थानस्थो विवर्धते ।
स ऊर्ध्वं कासवेगेन बहुरूपः प्रवर्तते ॥
जायते व्याधयश्चातः षडेकादशधा पुनः ।
....
कासोऽसतापो वैस्वर्यं ज्वरः पार्श्वशिरोरुजौ ।
शोणितश्लेष्मणोश्छर्दिः श्वासः कोष्ठामयोऽरुचिः ॥
पिच्छिलं बहुलं विस्रं हरितं श्वेतपीतकम् ।
कासमानो रसं यक्ष्मी निष्ठीवति कफानुगम् ॥

अंसपार्श्वाभितापश्च तापः पादकरस्य च ।
ज्वरः सर्वाङ्गश्चेति लक्षणं राजयक्ष्मणः ॥
अतिखिन्ने शरीरे तु यक्ष्मिणोः विषमाशनात् ।
कण्ठात्प्रवर्तते रक्तं श्लेष्मा चोत्क्लिष्टसंचितः ॥

Ibid. 40, 43, 49, 50, 55.

"The srothas being obstructed, the rasa remains in its own place. Accumulating there it is expelled by the urging of cough in various forms. From this state of things eleven ailments spring constituting yakshma. They are as follows:—Cough, pains in the shoulders, hoarseness of voice, fever, headache, pains in the flanks, vomitting of blood, expectoration of phlegm, breathlessness (Swasa), loose motions, and disgust for food...... The kapha expectorated while coughing is slimy, copious, smelling raw meat, green in colour or white or yellow. Pain in the shoulders and sides, burning of the palms and soles, and fever extending over the whole of the body together, constitutes the cardinal symptoms of the disease...... In a person afflicted with Kshaya the body becomes exceedingly lean through inharmonious food and drink, then blood comes out of his throat or phlegm which has been accumulating and which separated from the uras has a tendency to come out."

From the aforesaid facts pertaining to the combination of various causes subjective and objective which operate on human beings and bring about the disease are lowering of the resistance on vitality. This is same as modern workers speaking of disposition as that peculiarity in the organism, which allows for the effective working of the exciting causes of the disease. Disposition or casuation may be acquired or inherited. *According to Ribbet* "*acquired disposition may arise through a lowering of the body forces.*"

The above mentioned etiological factors both intrinsic and extrinsic and the lakshnas of the disease thus manifesting, give a very clear picture of the disease. But one will miss the krimi factor which was made very much of up till very recently in Europe, not having been discussed in the chapters concerning yakshma nor do they seem to have been given any place of importance. Unlike the ancient European medicine Ayurveda did recognize the existence of disease causing germs, both microscopic (adrusya) and macroscopic (drusya). Four varieties are described, *viz.*, (1) those born of faeces (Malaja), (2) those born of kapha (Kaphaja), (3) those born of blood (Rakthaja) and (4) those born of impurities. To us those born of kapha and blood are of some importance. Charakacharya [22] describes them in the following terms :—(1) "Of the krimies (bacteria) born of blood, the origin is the same as those of several varieties of Kushta (Leprosy). As regards their form they are very minute, round and feetless (limbless). Owing to their minuteness some of them are invisible. Their colour is coppery. The effect they produce are destruction of the skin, srothas, tendens, flesh and cartilage (Tharunasthi)".

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[22] शोणितजानां तु खलु कुष्ठैः समानं समुत्थानम्, स्थानम्, रक्तवाहिन्योधमन्यः । संस्थानं अणवो वृत्ताश्चापादाश्च सूक्ष्मत्वाच्चैके भवन्त्यदृश्यः । वर्णः ताम्रः । प्रभावात् ....त्वक्शिरास्नायुमांसतरुणास्थिभक्षणमिति ।

Charaka Vimana VII, 11.

(2) "The germs that are born of phlegm have their origin in the use as food and drinks of milk, raw sugar, sesame seeds, fish, flesh of animals living in marshy regions, food taken before digestion has occurred, food consisting of rotten substances, moist food, food prepared by mixing diverse articles, food composed of incompatible ingredients, and food that is not agreeable to the constitution. Their habitat is amasya. When they become powerful they move upwards or downwards or move bothwards. There are diversities with regards their form and colour. Some are white and some have forms resembling flesh (or muscles) some are like earthworms, *some are partly white and some are partly coppery*; *some that are white are minute, long and threadlike.* They are significantly enough known as antharada, udarada, and hirudhayada. The effects they produce are hrullasa, discharges from the mouth, disgust for food (arochakam), indigestion, fever, swoons, yawns, sneezing, anaha, pain all over the body, vomitting, leanness, and roughness of the skin."

श्लेष्मजाः क्षीरगुडतिलमत्स्यानूपमांसपिष्टान्नकुसुम्भस्नेहाजीर्ण पूतिक्लिन्न-संकीर्णविरुद्धासात्म्यभोजनसमुत्थानाः तेषामामाशयः स्थानम्। प्रवर्धमानास्तूर्ध्वमधो वा विसर्पन्त्युभयतो वा । संस्थानवर्णविशेषास्तु श्वेताः पृथुब्रध्नसंस्थानाः केचित्, केचिद्वृत्तपरिणाहा गण्डूपदाकृतयश्च श्वेताः ताम्रावभासाः केचिदणवोदीर्घास्तन्त्वाकृतयः श्वेताः तेषां त्रिविधानां श्लेष्मनिमित्तानां क्रिमीणां नामानि उदरादा अन्त्रादा हृदयचराश्चुरवो........इति । प्रभावम् हृल्लास आस्यस्रवणमरोचकाविपाकौ ज्वरो मूर्च्छा जृम्भाक्षवथुरानाहो अङ्गमर्दच्छर्दिः कार्श्यं पारुष्यमिति ।

Charaka Vimana VII; 12.

The abovementioned description of the varieties of krimis (bacteria ?) present a striking resemblance to the bacteria discovered by Koch, both in their shape and the conditions they cause. The krimies have not been viewed as separate entities operating immediately they find a lodgement in the body by producing diseased conditions. They have to be looked at in the background of the human body, with all the protective forces inherent in it. The mere presence of krimies alone will fail to cause morbidities. They always need circumstances favourable for their healthy growth and conducive to the casuation of specific conditions. Their habitat by whatever source they may get there as the blood-bearing vessels and amasaya, only suggests what is known in modern times as hematogenous and lymphogenous modes of dissemination. The proper culture media for them to grow and cause mischief are the unexcreted malas consequent to the disturbance and impairment of the successive parinamas of food as rasa, raktha, mamsa, etc. This is so particularly in the lungs. Speaking of this Prof. Osler has summarized the position in the form of an interesting allusion "Some seeds fell by the wayside and the fouls of the air devoured them up." These are the bacilli scattered broadcast outside the body, of them an immense majority die. "Some fell upon stony places". These are the bacilli that find lodgement in many of us perhaps with the production of a small focus but nothing came of it, they wither away because they have no root. "Some fell among thorns and the thorns sprang up and choked them." This represents the cases of tuberculosis, latent or active, in which the seed finds the soil suitable and grows, but the conditions are not favourable, as the thorns representing the protecting forces of the body, getting the better in the struggle. But others fell on good ground and sprang up and bare fruit a hundredfold.'

Regarding the communicability of the disease Ayurveda thinks in the same way as Western medicine, as will be evidenced by the following, quoted from Madhava Nidhana. ' By speaking, contact between bodies, inhaling, dining together, sleeping together in the same bed, using the same cloth, garlands and using the lepam used by others, leprosy, fevers, sosha, conjuctivitis, diseases known as aupasargika rogas travel from man to man.'

प्रसङ्गाद्गात्रसंस्पर्शान्निःश्वासात्सहभोजनात् ।
एकशय्यासनाच्चैव वस्त्रमाल्यानुलेपनात् ॥
कुष्ठं ज्वरश्च शोषश्च नेत्राभिष्यन्द एव च ।
औपसर्गिकरोगाश्च संक्रामन्ति नरान्नरम् ॥

Madhava Nidana, IX ; 40, 41.

We may therefore add the presence of the bacilli or krimi as one of the important symptoms of the disease and not as a primi cause, bearing of course in mind that the tuberculous disease must be preceeded by infection but infection need not always be followed by disease. Regarding the pathology of the disease, the term, Urakshatha and Vranora are significant. The ancient acharyas have very carefully understood the extensive ulceration caused in the viscera of the chest *viz.*, puppusa or lungs.

A very important factor which strikes me as being common between Hindu Medicine and Modern Medicine in phthisiogenesis is the importance of the gastro-intestinal tract (Amasaya and Pakvasaya) and the lymphatic system (Rasavahani Srothamsi) as a whole. The argument of the votaries of Hindu Medicine is that long before the disease manifests with all its classical symptoms, appetite becomes less, digestion impaired, absorption and assimilation faulty, consequently the patient grows thinner in spite of feeding him with the best of chosen food. They also think that with the vitiation of Rasa (or lymph?) all the other Dhatus like blood, muscle, bones, etc., fail to get their proper quantum of nutrition, the lymph channels becoming involved in the process of the disease. There is hardly anything in this to be disputed. It is only very much later on, that other organs like lungs show signs of involvement and it is only a symptom-complex of a chain of disturbances having their origin in the gastro-intestinal tract and the consequent dislocation of the successive parinamas of food from Rasa to Ojas, due to organic and functional disturbances in the tract itself.

It is not only in our experience but also in that of great workers in America and Europe, that no one given Therapy is by itself enough in the treatment of the disease. The disease is **a local manifestation of a general disturbance**, and a successful therapy must be one, which should combine not only the treatment of the part but also the primary errors due to which the disease usually manifests as a localized affection, as for instance in the lungs. From this it follows that the primary condition *viz.*, the primary errors (functional and organic) beginning from the gastro-intestinal tract, should be firstly corrected, to help the body to derive its energy, not only to fight the disease but also to repair the wounds. This particular aspect of the question has escaped the attention of people who are in charge of institutions of Tuberculosis in our country. While in Europs and America treatment of the lung with the collapse therapy has been relegated to two to five per cent of the total number of cases, in our country it is being adopted in almost all cases. In this connection it will be relevant to quote from Fishberg the following.

[23] "Proportion of cases suitable for therapeutic Pneumothorax :— 'From a study of indications and contra-indications, it appears that relatively few patients have lesions that lend themselves to be benefited by therapeutic pneumothorax. Most, who are competent to speak authoritatively find that less than 5 per cent. of all active pulmonary tuberculosis that come under their care in private and institutional practice can be considered ideal for pneumothorax. For over twenty years I have found less than 5 per cent. of cases in which I would conscientiously urge this form of treatment, and of these, at least ⅓ had sufficient pleural adhesions to render the attempted collapsing of the lung more or less futile. Bernard (Paris) found among 628 patients only 22 in whom he thought pneumothorax is indicated and among these he succeeded only in 6 cases to collapse the lung completely and in 11 adhesions prevented the creation of an active pneumothorax.'

'The Mastons, reports (Am. Rev. Tuberculosis, 1929, 322) that 600 of 7000 patients or 8·5 per cent. were considered suitable. Of these 120, or 40·8 per cent. the collapse attained was only partial and only in 235 or 39·2 per cent. was satisfactory pneumothorax produced.'

[24] *Limitations of Pneumothorax Therapy* :—' 'It is unfortunate that only in a relatively very small proportion of cases of pulmonary tuberculosis is this method of treatment appropriate. The most sanguine advocates of pneumothorax, who would operate on patients in whom the indications are questioned by other authoritative clinicians, find, that hardly 5 per cent of all tuberculosis cases are suitable for the treatment. I am inclined to agree with those who hold that in less than 1 per cent of the total humber of persons with active tuberculous disease of the lung is pneumothorax indicated. It is thus obvious that therapautic problem of the disease have by far, not been solved by this method of treatment. It is undoubtedly a boon for many an individual patient, and it has saved thousands of men and women who would have succumbed to the disease without artificial pneumothorax, but over 95 per cent of all who suffer as a result of tuberculous infection must still be cared for along other lines of treatment.

We have shown that in the cases on which the treatment is applied, the results may be encouraging, even brilliant. The cardinal symptoms of tuberculous toxaemia are ameliorated, the cough is lessened, the amount of sputum reduced to a minimum, haemorrhage stopped etc., and the patient gains in weight and strength. With no other available method of treatment have we attained these salutary results as are evident in *certain selected cases,* (Italics mine).

It is however, unfortunate that in a large proportion of treated cases the attained improvement is not permanent and lasting. Within a few weeks or months, with or without any obvious reason, there occurs an unfavourable turn and the patient begins to slide downhill. It is often disheartening in a case that has been collapsed, some complication undoes all the salutary clinical results within a few days. In upward of 50 per cent of the cases, pleurisy, serous or purulent occurs, which often compels the abandonment of the treatment and the patient is no better, often much worse, than before the induction of pneumothorax. To be sure, some patients are not harmed by a small, at times, even by a copious effusion, but in the majority, it is inimical to recovery. In about 26 per cent a lesion in the contralateral lung flares up and nullifies the beneficial effects of the collapse of its mate. In still others, extra-thoracic tuberculous lesion especially of the larynx and intestines made their appearance and the ultimate outlook becomes gloomy indeed. In a large proportion of treated cases, a satisfactory collapse of the diseased lung cannot be obtained because of pleural adhesions and even the temporary improvement noted in others is not impressive or is altogether lacking. It is the experience of those who have treated numerous patients that satisfactory collapse is obtained in less than 50 per cent of selected cases.

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[23] M. Fishberg Pul. Tuber. Vol. II, P, 452-53.

[24] *Ibid*—Vol. II, P. 553, 554 and 555.

As shown above, less than 5 per cent of patients with active tuberculosis are suitable for treatment and in the majority of these a favourable clinical effect may be expected, but actual lasting therapeutic results, cicatrization of the lesion, does not occur in more than 2 per cent of the treated cases. In other words, therapeutic pneumothorax cannot be considered a universal therapeutic measure for the disease; it is not as effective in salutary results as the operations for appendicitis, cholecystitis, gastric ulcer etc., in which there are some drawbacks, still we can expect favourable ultimate results in 95 per cent of the cases operated upon. At best, lasting results are obtained in less than 25 per cent of cases treated with pneumothorax.

Tuberculosis is a very costly disease; when treated with pneumothorax it is even more costlier. While in some successful cases the treatment may be terminated in little less than one year, this is exceptional. In the majority, refills have to be made for two to three years and in some for more than five."

Fishberg adds ' that it is obvious that therapeutic problems of the disease have by far not been solved by this method of treatment '[25].

If this is the argument for a comparatively simple measure as pneumothorax, it needs very little imagination to contemplate the possibility or the advisability of other major surgical interferences like phrenicectomy and Thoracoplasty. This is not to mean that we are blind to the value of these paliatives aspects of treatments but **what is primarily required is the treatment of the disease in its entirety and not of a part alone.**

In passing, a word about the therapy with tuberculin which is still in vogue in our country needs a mention here. ' The so called specific treatment, the various tuberculins, sera and vaccines, which have been hailed for their alleged curative powers when properly administered, are not stated to have any known bactericidal action, nor are they known to hinder the proliferation of the bacilli within the body or to immunise the tissues against the poisons engendered by these micro-organisms through the production of antibodies, as is the case with antitoxins. Attempts at active or passive immunity have not met with notable success in tuberculosis '.[26]

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[25] M. Fishberg. Pul. Tuber. Vol. 2, pages 553—55.

[26] *Ibid*—p. 268.

He further says that 'there is no evidence in medical literature that any investigator has succeeded in curing or benefiting a tuberculous animal with tuberculin treatment and what has been observed however, is that very often dormant tuberculous process are activated after its administration. Bacilli which gave no trouble were released, 'mobilized, producing a bacterimia.'[27]

In this connection it will be useful to bear in mind that where we are unable to understand and interpret the mysterious warnings sounded by Dame Nature, the best service we can possibly render unto her will be to refrain from acts of vandalism on Her Sacred preserves, unless there is an imperative need warranting such an act.

Coming to other measures, practically almost all the drugs which held the field at one time or other like iodine, calcium, copper, arsenic, creosote etc., had their own limitations and hence are almost abandoned. However it will be relevant to mention here of the injectable Gold compounds which held the field in Europe till recently and continues to hold the field now in our country. The basic principles on which injectable gold compounds were developed in Europe were their bactericidal action on the tubercle bacilli in dilution of 1 in 2,00,000 *in vitro*. This was tried by Robert Koch in 1890 in the form of Gold Cyanide. It was given an extensive trial in Europe, in human patients and finally abandoned as useless in tuberculous disease in human beings.

The next preparation was that of Mollgard who built a compound of gold (gold thiosulphate) naming it Sanocrysin. As in the case of Cyanide of Gold, Sanocrysin was also capable of destroying the bacilli in vitro. He claimed that it was less toxic and reported optimum effects. This preparation was found to be more suitable in cases with exudative lesions without fever or almost with mild subfebrile temperature. It was administrated intravenously. Various disturbing reactions were observed in a large proportion of cases during the treatment, indicating that it is not a safe remedy. Hiccough and vomitting were found to follow gastric derangement and profuse diarrhoea have been observed in many. Skin eruptions were not rare during the treatment The establishment of albuminuria was noted and in some instances the shock and collapse immediately after the injection proved very serious and even ended fatally. After a fair trial in various parts of the world except in India, Sanocrysin has been abandoned in Phthisiotherapy by nearly all of its enthusiastic advocates.

This once again brings us back to what Indian Medicine actually offers. It reiterates that the disease is not exclusively of bacterial origin, that it is due to the reaction of the subject to the errors in the correlation of Ahara, Viharas and perversities of Kala; that the primary trouble commences long before the pulmonary lesion develops in the gastro-intestinal tract and in the lymphatic system, and that all measures must be aimed at, to the restoration of Jataragni in the gastro-intestinal tract as well as the Dhatvagni. Without these primary conditions being satisfied, all other measures like prolonged stay in a sanatorium, absolute rest in bed, vary nutritious food abounding in all the principles of diet including Vitamins from A to Z, God's good fresh air, and at the top of all these surgical collapse therapy will be of very little value. This is not to mean that we claim very much for drug treatment either. **All things being equal, a**

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[27] M. Fishberg Pul. Tuber, Vol. II, p. 268.

drug which aims at the correction of the primary errors referred to above will be useful.

It looks as though the views of eminent workers as M. Fishberg are the best commentary to the ideas held by those of Hindu Medicine. He says that 'The aeteology of tuberculosis however teaches a lesson in rational therapeutics. The tubercle bacilli do not grow with equal facility in every individual; if they did, the number of human beings who succumb to this disease would be equivalent to the number that give positive reactions to tuberculin, indicating that they have been infected with tubercle bacilli; over 90 per cent of the adult population in large urban centres! In what this favourable soil consists, we are not altogether clear. In the chapter on Phthisiogenesis we discussed it in detail, and it was *evident that everything which undermines the general health of a person and reduces his vitality may prepare a favourable soil for the growth of tubercle bacilli within the body and thus produce phthisis. As a corollary we may agree that anything which will stimulate the vital defensive forces, which are more or less inherent in every individual or which will improve the nutrition of the body, may hinder the proliferation of the bacilli and with the improvement in the general physical condition of the patient the local lesion may cicatrize or* **the dissemination of the bacilli by metastasis may be prevented.**[28]

The failure of most of allopathic therapies are probably due to attempting to correct the bacteriological factors rather than the constitutional, which are conducive for the bacilli to become menacing. There is always a difference between an attempt to reach the pathological lesion through an unnatural route, as for instance, through the vein on one hand and through the natural route, viz., the oral route equipped at every successive stage with protective and counter-acting apparatus like liver on the other. This is more so in the case of the administration of heavy metals. Needless it is to say that powerfully bactericidal (in vitro) substances like Sanocrysin, are unable to penetrate the tubercular giant cell system to be able to destroy the bacilli *in vivo.*

May I beg of you to forgive me for this digression, if it be a digression, in view of the fact that without a comparison it is difficult to assess the value of different therapies. Gold in Indian Medicine has been esteemed as a metal with a preponderance of 'Thejobhuta', capable of correcting the errors which occur in the Dhathus and therefore the Dhoshas.

न सज्जते हेमपांगे पद्मपत्रेंऽवुवद्विषम् ।
जायते विपुलं चायुर्गरेप्येष विधिः स्मृतः ॥

Ash. Hru. Suthra. VII, 28.

It is esteemed as very valuable in neutralising toxins of vegetable and bacterial origin. It is esteemed as valuable in chronic toxic inflammatory states. It forms an important part in the composition of good many medicines indicated for wasting diseases like Rajayakshma.

One such gold preparation 'Calcii-Aurum' (Swarna-Pravala) prepared according to traditional Indian methods was subjected to pharmacological animal experiments at the Faculty of Medicine, Hamburg

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28. M. Fishberg, Vol. II. Pul. Tuber. pp. 268, 269.

University, Germany, by the author and the following results were noted:

1. That the gold in the compound is absorbed when given by mouth.
2. It is deposited in the reticulo-endothelial system.
3. It is not toxic as other gold preparation of modern medicine are.
4. It possesses both Organotropic and Bacteriotropic properties, and its beneficial action is due to its oligodynamic properties.
5. It is capable of increasing the resistance of the animal organism against bacterial infections.
6. In tuberculous subjects it ensures an increased appetite and improves a general sense of subjective well-being. Definite controlling effect on the temperature and equally good increase in weight, abolition of night sweats, lessening of cough and easy expectoration are observed.
7. Accelerating the healing process in the tubercular lung with proliferative change including fibrosis was noted. (These fullfil the requirements specified by Maurice Fishberg quoted elsewhere.)[29]

This preparation forms the bedrock of our therapy for tuberculosis in the Government Indian Medical School Hospital. At the first instance it was decided to admit only advanced cases wherein none of the surgical collapse measures could be done due to the bilateral nature of the lesion with plenty of adhesions and wherein injectable gold treatment has been tried and abandoned.

An important aspect of the treatment is its individualisation in its details to suit the cases. They are elastic and adaptable to the polymorphous nature of the disease, with due regard paid to the various accidents and complications occurring during its course.

The object of admitting fairly advanced and bad types of cases was not with the intention of curing them but to observe how far the use of this traditional Indian Medicine along with the classical Santharpana Chikitsa according to Ayurveda is capable of arresting the rapidly progressive disease.

Santharpana and Vathathapika Chikitsa are the mainstay of the treatment contemplated for the disease by Ayurveda. These conform to the rest and sanatorium therapy elaborated by Western medicine. Talking of Santharpana Chikitsa and indication for it, Vagahata says as follows:—

"Santharpana Chikitsa should be done when either vatha or vathapitha is vitiated; this treatment constitutes a samana therapy. Those who are afflicted with different diseases; those in whom different kinds of medicines are administered; those who are addicted to drink; those who have dissipated themselves by excessive sexual pleasure; those who have become lean on account of anxieties; those who have borne heavy burdens; those who had to walk over much; those who have lost their health and vitality due *to Urakshatha*; those whose bodies have become dry and weak and in whom vatha is predominant, pregnant women, parturient women, boys, old people are fit persons for Sanatharpana Chikitsa".

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29. "Experimental Investigation over the Pharmacological and Therapeutic properties of calcii-Aurum"—Antiseptic, Jan. 1941.

616.24613 9√632

बृंहणं शमनं त्वेन वायोः पित्तानिलस्य च ।
बृंहयेद्व्याधिभैषज्यमद्यस्त्रिशोककर्शितान् ॥
भाराध्वोरः क्षतक्षीणरूक्षदुर्बलवातलान् ।
गर्भिणीसूतिकाबालवृद्धान् ग्रीष्मेऽपरानपि ॥

Ashta. Suthra XIV ; 7–8.

"One whose body is wasted, when living without anxieties, mind permeated with joy, treated with santharpana and having adequate sleep, will soon become fatty like a varaha".

अचिंतया हर्षणेन ध्रुवं संतर्पणेन च ।
स्वप्नप्रसंगाच्च कृशो वराह इव पुष्यति ॥

Ibid 34.

There was another object behind this choice of cases, *viz.*, the kind of criticism which may be levelled against us, in case we started with fairly early cases. Out of the 17 cases treated so far, within the last 3 years, about 8 cases including 2 early cases responded very well to the treatment as will be seen from the X-Ray pictures and weight curves.[30] Two of the 17 died, and 7 patients did not undergo the full course of treatment having got themselves discharged within 7 to 10 days after their admission. In all these cases the appetite was found to be voracious and the temperature curve fell inversely to the weight-curve. Their cough became less and in most of them occasional unproductive dry cough was found to linger. A perusal of the serial skiagrams will show an arrest in what was progressively an advancing disease, with proliferative fibrotic changes. These patients were all given a very rich diet which comprises of 6 boiled eggs, 3 to 4 pints of milk, 12 oz. of tomatoes, 6 oz. of honey, one pint of ragi kanjee, rice, ghee, vegetables, mutton soup in the case of non-vegetarians, almonds 4 oz. bread 12, butter 6 oz. and buttermilk.

This fabulous diet will, I think, in the case of a normal man, be enough to cause diarrhoea and colic for several days but these fairly advanced pulmonary tuberculosis patients were still in need of more food.

The patients are ordered to complete rest in bed and accommodated in a big shed attached to the ward in the Ayurveda compound. This enables them to get plenty of fresh air. The other medicines which form an important adjunct in the treatment are more or less symptomatic. As a routine, the following are prescribed :—

(1) Bhoonimbagudoochi Kashaya two oz. three times daily with a teaspoonful of honey added to each dose. The Kashaya is made as follows :—

*(*a*) Boonimba	6 tolas
(*b*) Gudoochi	4 ,,
(*c*) Vasa	4 ,,
(*d*) Kantakari	4 ,,
(*e*) Thulasi	6 ,,
(*f*) Mustha	4 ,,
(*g*) Sunti	5 ,,

पुस्तकालय गुरुकुल कांगड़ी

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* "Samoolam."

[30] Refer to the Indian Medical School Magazine, January 1942.

Add a measure of water; boil to 1/8; of this two ozs. each time is to be administrated.

(2) Bhaskara-lavana Grs. 15 for each dose, thrice daily with masthu or warm water after each meals, morning, noon and evening.

(3) Akara-karabadhi vataka 3, each pill to be kept in the mouth and the juice sucked. Its composition is as follows:—

Akara-kara choornam	5 grs.
Sunti choornam	1 ,,
Pippalee choornam	1 ,,
Yashti choornam	3 ,,
Yashti Niryasam	2 ,,
Rasna	1 ,,
Ahipenam	1 ,,
Apahvakarpoora	1 ,,

The following is the method of preparing large numbers of the Akara-karabadhi pills. Multiply the quantity of drugs by the number of pills required and the total quantity of the powder should be rubbed at the rate of 6 hours per day for three days with fresh vasa swarsam (samoolam) and rubbed similarly for three days more with Kantakari Swarasam (samoolam), again with Thulasi Swarasam likewise, finally with enough honey until the whole mass becomes fit enough to be made into vatakas. The vatakas are to be dried in diffuse sunlight and preserved in airtight bottles.

(4) Chyavanaprasha avalebam:—1 tola each time, twice daily, is given in the morning and evening.

(5) In cases of haemoptysis or Rakthashteevanam, the following is prescribed:—

Pravala	5 grs.
Laksha	10 grs.

Such 4 to 6 powders should be given in 24 hours with honey. More often this condition clears up within one or two days. Sometimes when haemoptysis is of an alarming nature, the pravala medication is supplemented by intravenous injection of calcium gluconate (Sandoz 10 per cent.) 10 cc. along with 1 cc of celin (Glaxo). This is given almost every day and sometimes twice a day depending upon the nature of haemoptysis. The uses of calcium gluconate, however, happened to be on rare occasions and in few cases.

(6) Sometimes when the patient is very much disturbed by paroxysmal coughs, syrup codiene one drachm is added to each dose of the Boonimba-gudoochi kashayam.

(7) The use of morphine was resorted to once or twice in some of the cases which had haemoptysis. These occasions were, however, few.

Conclusion:

From a study of the case reports of the number of cases treated at the Indian Medical School Hospital and their serial skiagrams it will be clearly seen that the main treatment for the disease pulomnary tuberculosis should be aimed at to restore the Dhosha equilibrium and to increase the Jataragni and Dhatwagni. When this is done the body will be able to utilise the nourishment supplied to it. This alone is capable of contending against the rapidly progressive disease. In other words, errors organic and functional endangering the successive parinamas of food from Rasadathu to Ojas should be corrected. This is achieved best by the administration of the special Swarna Pravala preparation.

The cases treated as already mentioned were of very advanced types wherein a total cure was not to be anticipated except in the case of one. In all the pulmonary types modern collapse therapy was contraindicated on account of the extensive bilateral nature of the disease and adhesions.

It has been clearly brought out that the collapse therapy is useful in only 2 to 5 per cent of cases of pulmonary tuberculosis and that the rest will have to be treated by other methods. Our claim based on the number of cases we have so far treated is that the Indian Auro-therapy per os. is probably the one, which as compared to others, does much good. In less advanced cases, the therapy is naturally expected to be much more beneficial. The author however feels that collapse therapy by itself will not be sufficient even in those cases where it is indicated, as long as the pulmonary manifestations of the disease happen to be a *local manifestation of a general disturbance*. Unless the nature of the general disturbance is understood and corrected, the local trouble with collapse therapy will ultimately prove itself to be only of transient value. The author personally feels that local treatment to the lungs with collapse therapy in cases having all the indication for it, can be treated with great advantage by combining the Indian gold therapy per os.

One very relevant question may be asked as to why the tubercle bacilli have not totally disappeared from the sputum of some of the patients. This is answered best by the fact that so far no therapeutic measure has been successful in destroying the parasite *in toto* within the body. This applies to both medicinal and collapse therapies. What has however happened in almost all the cases wherein clinical healing or arrest or quiescence has taken place is that the activity of the bacteria have been checked by the increased resistance of the patient. The disappearance of the bacilli from the sputum is at best an evidence of their being held secure by the healing process. But this never assures against a future re-infection or super-infection or opening up of the healed lesions, which however depend upon how the patient lives. Hence the appearance of stray bacilli, when the clinical condition, as for instance fever, cough, expectoration, dyspnoea, tachycardia, insomnia, night sweats, impaired appetite and digestion, anorexia, emaciation and loss of weight have all been abolished and when this state persists for periods exceeding three months, it is almost the same as harbouring the organism within the lungs, though kept under restraint. The factors, which after all have to contend against the ravages of the disease, bacterial or otherwise, are those which are inherent in the body and this to a very large extent depends upon proper nutrition and its utilisation by the body. When nutrition is properly utilised and converted into various constituent elements of the body, supplying the most immediate needs due to wear and tear but also replenishing the reserves, it will be found that the disease process wanes inversely. The body soil or Karyayoni then becomes an unsuitable media for the bacteria, which are forced to recede. If this state of affair is not only brought about but also maintained, the appearance of a few bacilli here and there in the subject's sputum ceases to be of importance. It is even possible that these bacilli may totally disappear ultimately if this condition is maintained for long periods Their growth and spread, causing further damages to the part involved will be controlled by the altered environment of the body soil and restored fluid conditions. A culture test in such of those sputums which exhibit no bacilli by direct methods as Zeil Neelsen or anti-formin, is fallacious as the ideal medium and temperature supplied to the bacilli outside the subject's body strictly excludes the various factors which contend against them *in vivo*, in respect of its growth, activity and capacity to cause morbidity. In support of this statement we may quote Theobald Smith. [31]'To-day we know that to have identified the microbic agent of any pathological process is but the beginning of the solution and that it answers one of a long series of questions......*non-parasitic factors are necessary conditions in the origin of infectious diseases and far outweigh the living agent in aeteological significance.*' [32]'Maurice Fishberg may also be quoted to this end when he says that 'Attention has recently been turned to accessory non-parasitic causes of phthisis, among which there are many including endogenous, such as heredity, anatomical and *biochemical peculiarities* (italics mine) of the individual etc., and also exogenous, including environmental peculiarities, such as economic conditions including occupation, housing, nourishment etc., and also geographical millieu.' Finally let me conclude this paper with another quotation from the pen of the same authority. [33]'So long as there is persistently but little cough, or none at all, no fever, no tachycardia, dysponea, chills, sweats, etc., the prognosis is good no matter what physical exploration discloses. *Continuous freedom from these symptoms for several months is an indication of arrest even if tubercle bacilli are found in the sputum*'.

These views are not quite unfamiliar to us but are almost identical with what has been said in the ancient books on Indian Medicine.

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[31] Science, 1921, N.S., Vol 54, 99.
[32] M. Fishberg, Pul. Tubercle, Vol. I, p. 115.
[33] Ibid Vol. II, p. 247.

Liberty Press, Madras.

Issue No. 2

TRANSACTIONS
of
The Academy of Indian Medicine
MADRAS

(1941—'42)

'Abdominal pains in relation to Food'
(PART I)

By

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Price Annas Twelve.

ABDOMINAL PAINS IN RELATION TO FOOD

INTRODUCTION :—

Sometime back, an announcement was made by the medical research unit of Government of India, to the effect that a great many people in Southern India suffer from peptic ulcers. It was also suggested, that the main cause for such incidence in Southern India was probably the lack of consumption of cereals. An attempt was made to verify that statement in the Indian Medical School Hospital, Madras, on a small scale. About 10 per cent of the cases admitted was for peptic ulcers.

For our purpose of study it is necessary to understand the Ayurvedic conception of certain of the abdominal pains. This paper would confine itself to such of the abdominal pains which are related to food and so, the pains that arise due to organic or functional disorders of the alimentary canal—*maha srotas*. In other words it is intended to discuss the ætiology, symptomatology, diagnoses and treatment of *Sulam* and *Gulma*.

HISTORY :—

Before entering into the subject proper, a brief history of the disease would be useful. In Charaka Samhita, which is the earliest of the medical books available (6th Cen. B. C.) no mention is made of Sulam as a disease. It recognises Gulma but not Sulam as a separate entity. Sulam is considered as one of main preliminary symptoms of Gulma. In Sushruta Samhita (3rd Cen. B. C.) Gulma is separately taken up and Sulam separately but, the classification of Sulam is different from the later books. In Ashtanga Sangraha and Hridaya (2nd Cen. A. D.) no separate mention is made of Sulam. So it may be presumed, that the Atreya school of thinkers thought, that Sulam need not be classified as an entity but only as a prodomal symptom to Gulma (कुरुते शूलपूर्वकम्) or as a main symptom of Gulma, where as the Dhanvantereyas thought that Sulam should be classified as a separate disease.

From the time of Madhavakara onwards (8th Cen. A.D.) to that of Bhava Misra (16th Cen.) every author has classified the disease separately and also enlarged the classification and treatment of Sulam. Perhaps, as the days advanced, people got into unhealthy habits and consequently suffered more from the abdominal pains to necessitate a separate classification.

DEFINITION OF SULAM :—

शंकुस्फोटनवत्तस्य यस्यात्तीव्रवेदनाः ।
शूलासक्तस्य लक्ष्यन्ते तस्माच्छूलमिहोच्यते ॥

Sus. Utta. Ch. 42, Sl. 81

The word meaning of Sulam is shooting pain as though pierced with sharp instrument and it is always due to the vitiation of vayu. This pain may come anywhere in the body; but as a specific (Rudhi) name it always indicates pain in the abdomen due to disease of the digestive tract. Two general classifications are made by Madhavakara, namely, Sulam, pain in the abdomen and Parinama Sulam, pain in the abdomen during digestion (anna Parinama). In this classification all other pains arising out of granthi, apachi, valmika etc., various benign and malignant tumours in the alimentary tract are excluded.

VARIETIES :—

We shall now take Sulam in general. This is of eight varieties, three caused by the vitiation of individual doshas, three by the vitiation of two doshas (dvanda) one by the vitiation of all the doshas and one by Amadosha. In all the varieties of Sulam Vayu is the predominating dosha.

सर्वेष्वेतेषु शूलेषु प्रायेण पवनः प्रभुः ।

Mad. Nid. 25–1.

CAUSES :—

General causes mentioned under the causation of this disease are: excessive physical exercise, riding, sex indulgence, loss of sleep, drinking very cold water, eating foodstuffs that are dry, trauma, suppressing the bodily urges as stools, urine, flatus etc., sorrow, fasting and speaking or laughing excessively. When Vayu gets vitiated by these ways, the other doshas get vitiated by causes which increase them and both combining cause pain.

SIGNS AND SYMPTOMS :—

Along with the general symptoms of vata[1] in this disease, when vata is predominent pain comes at the end of digestion, in the evenings and in rainy season. Drinks and foods which are cold or of sitha virya aggravate the condition and the pain is relieved by fomentation, application of oils and taking in warm oily foods. In the case of pitta sulam along the general symptoms due to the vitiation of Pitta, the pain comes during the midday, midnights, during the first part of digestion (Vidaha Kala) and during Sarat kala. It gets relieved by sitoparcharam and by taking food which is of sweet taste and sitavirya. Along with the other general symptoms that are produced with kapha, the pain in the abdomen starts immediately after food, early in the morning at the sun rise, in winter and autumn seasons. The pain would be relieved by warm and dry applications and food with the same attributes. In dvanda and sannipata varieties the signs and symptoms of the combined doshas would appear. Most of these pains are correlated as gastritis or gastric ulcers. In Madhava Nidanam another kind of Sulam is mentioned and that is parinama sulam. In this disease all the three doshas get vitiated. When the vitiated vayu gets obstructed by kapha and pitta, pain is caused. This pain comes in the later part of digestion. The pain is felt on the sides of the abdomen over the unbilicus in the hypogastric or epigastric regions. It may also be felt over the back. The pain is relieved after food, or after vomitting or when the food is completely digested. Though this pain is of sannipata origin according to the preponderance of the doshas this is classified under seven varieties. This is correlated as deodenal ulcer.

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[1] A student of Ayurveda is expected to know the fundamentals of the doshas : when and how they get vitiated, the seasonal disturbances etc., so the details are not given here.

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From the modern point of view these classifications of Madhavakara are more intelligible than that of Sushruta's classifications. In these classifications we have the nature, the duration, the seat of pain and its relationship with food and the means to relieve it. Sushruta classifies only four varieties in the following way. Sulam due to vata, pitta, sleshma and sannipata. He also mentions Parsvasula due to vata kapha, Kukshi sula due to vata and ama, Hritsulam due to vitiation of vata and rasa, vasthi sulam due to vayu, Mutrasulam due to vayu, vitsulam again due to vayu. Somehow, in the diagnosis of pains in the abdomen, these classifications are not followed, at any rate, in this hospital. The classifications followed here are only in accordance with the classifications of Madhavakara. The correlations of these classifications with that of western medicine are given at the end of this paper. The classifications are in a study of 100 cases.

Now we shall take up the disease Gulma, which is mentioned in all the books and also treated as one of the important diseases. Gulma means a tumor. This also is caused by vitiated vayu. So both in Sulam and Gulma vayu plays an important role in the ætiology. Just as no fever is possible without the vitiation of pitta, Sula or Gulma is not possible without the vitiation of vayu. The other two doshas play a secondary role. In the causation of the disease it is important to note, that the disease comes only to such people who are already emaciated by any disease or who had done such acts and taken such diets which had made him thin. This ætiological factor is important to note; in Sulam the patient need not be emaciated, and in the course of the disease he may become emaciated, whereas, in Gulma, emaciation is a pre-requisite to the onset of the disease. When this emaciated person takes again foods and drinks, that excite vata or perform actions that vitiate it vayu gets vitiated.

यदा पुरुषो वातलो विशेषेण ज्वरवमनविरेचनातिसाराणामन्यतमेन
कर्शनेन कर्शितः........तस्यापचाराद्वातः प्रकोपमापद्यते ॥

Cha. Nid. III 6.

This vitiated vayu enters into the mahasrothas (alimentary canal) and becomes hard and rounded (Pindithakara) because of the dryness already existing there. This causes various kinds of pains and tumors. This rounded mass becomes bigger at times and smaller at other times in the case of Vatagulma. In Pittagulma along with the pain there will be burning sensation and tumor will be hot to touch and very tender. In Kapha gulma along with the general signs of kapha vitiation the tumor occupies Amasaya and causes pain. In Sannipata gulma all the signs and symptoms of the vitiated doshas would be present. The fifth which is Rakta gulma and comes only to women is out of the scope of this paper and so I leave it.

It may be clear from the above descriptions of gulma, the presence of tumor is an essential factor along with the pain and other symptoms connected with the vitiation of various doshas. This is a chronic process, as it has been mentioned above that it comes to those who are debilitated by various illness previously. In the case of Sulam, the person need not necessarily be lean and the onset may be sudden. These are the differences. They agree in certain respects. Though the pain may be referred all over the abdomen, it has got certain seats. They are Hrith (epigastric region) nabhi (umbilical region) Vasthi (Hypo gastric region) and two parsvas—the lumbar and hypochordiac regions on both sides. The nature of the pain and the relationship to food and the time of the day is the same both in gulma and sulam.

विनागुल्मेन यच्छूलं गुल्मस्थानेषु जायते ।
निदानं तस्य वक्ष्यामि रूपं च स चिकित्सितम् ॥

Sus. Utta. 42–76.

The seat and time of the pain in determined by the predominance of the particular dosha. That is during the day kapha predominates in the morning, 'pitta in the midday and vayu in the evening. The same order is also for the night. Immediately after taking food kapha predominates, during digestion pitta and at the end of digestion vata. When this principle is known it becomes easy to diagnose a case according to the dosha vitiation. So it may be concluded that in every case where there is a gulma, the pain is a concurrent symptom, and in the case of sulam no tumor is felt. Therefore in my opinion in the early part of the disease we can diagnose a case of sulam but when definite organic changes had taken place it becomes gulma.

In a study of 100 cases of abdominal pains treated in the Ayurvedic Wards, the following allopathic classifications are arrived at. The allopathic diagnosis for almost all these cases were arrived at with the aid of Skiagrams and the medical registrar, an allopathic medical graduate. The Ayurvedic diagnosis was made on the clinical findings of the case according to Panchalakshana Nidana. It may be seen from the tabular form given at the end, mostly gastritis and gastric ulcers go to form the diagnosis of sula put under various doshic headings and mostly the deodenal ulcers, pyloric ulcers go to form the parinama sula. It is interesting to note that description given above to parinama-sula agrees very favourably with the modern definition of deodenal ulcer. Another point is that more than one third of the cases are parinama-sulam. When pyloric stenosis has happened and when then is reverse peristilis, in diverticulties and such other diseases the diagnoses of gulma is made.

TREATMENT :—

As vayu takes an important place in the ætiology of the case and as generally people who are afflicted with sulam or gulma, have kroora koshtam, *i.e.*, constipated bowels, snehapanam plays an important role in the treatment. In cases where there is predominence of vata or pitta, ghritham is preferred and where kapha is predominant thailam is indicated. These svehanams are suitably medicated in accordance to the age of the patient, the avocation, season, and climatic conditions. Along with snehapana, externally various oils are applied and fomented. This is not done in the case of pitta-sulam and gulma, as it would aggravate the symptoms.

Another factor that should be remembered is the various medicines that increase the appetite. These medicines are given generally in powder form which contains various salts. The inclusion of salts and certain alkalies (kshara) is to neutralise the hyper-acidity as well as fermentation. When there is amlavipakam or vidagdhatha of the food stuffs, these ksharams convert them into madhura vipakam and so irritation of the affected parts is lessened.

Another most important treatment in sulam and gulma is vasthi—both asthapana and anuavasana. These are given to bring down the vitiation of vayu in its own place and the consequent derangement in other parts of the intestines. These also evacuate the bowels in the easiest way and tone up the walls of the maha-srothas.

Here, mention of certain calcium compounds as Sankam, Varatam and Sukti should be mentioned. It is interesting to note that in many of the preparations which are mentioned under the treatment of Sulam, iron is included in various forms as Dhathriloham, Sathavarimandooram, Taramandooram etc. In modern medicine also I understand absence of sufficient calcium salts in the blood and defective circulation are some of the causes for the peptic ulcers which is sought to be corrected by calicum and iron compounds.

I briefly mention here the medicines that were used in the wards according to their frequency, in the 100 cases mentioned above. The results of the cases treated are given in the tabular statement. Whether some of the cases that are put as 'relieved' or cured have not recurred is difficult to find out, as in India, the Hospital class of people are generally ignorant and illiterate. The follow up could not be done. If a patient is cured completely, he does not inform the doctor, if he is not cured he gets disgusted with the hospital and goes to another. In the city of Madras with number of hospitals, it is not difficult for him to find another.

SNEHANAMS :—

(1) Indukanta ghritham[1] takes the first place. This is given twice or thrice a day with hot water. (2) Satphala Ghritham takes the second place. (3) Sukumara ghritham, third place. The last mentioned is certainly better than the first two. But in prescribing in a hospital, the physician is also guided by the cost of the medicine, and so gets the third rank.

CHURNAMS AND KSHARAMS :—

Among the powders, Bhaskara Lavana takes the first place, the second is Ashta churna, the third place is given to Viaswanara churna. Among the ksharas, Sankavati gets the first rank, Varata bhasma, the second, Sanka bhasma third, Sukti bhasma fourth and Narikela lavana, fifth. In some of the cases, Vasthis are given along with one or other of the above mentioned medicines.

Recently, I had an opportunity to use a certain medicine called Taramandooram, mentioned in 'yogaratnakaram' under parinamasulam. It has given very good results, and I have also the authority of another physician, who has used it extensively in his practice and found very good results. As it is not in the hospital pharmacopoea there was no opportunity to have it tried here. I mention this so that the members of the Academy may give it a trial.

DIET :—

As this is primarily a disease of the digestive system, diet plays an important role. Diet which is nutritious, warm, oily and easily digestable

(Concluded on page 8)

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[1] Sahasra yoga ghrita Prakarana. Available only in Malayalam Script, a portion of it is printed in Nagaram Script. A Tamil translation also is available.

Allopathic diagnosis.	Indian diagnosis.	Doshic Classification.								Total.	Results.			Remarks.
		V	P	K	VK	VP	KP	VPK	Ama		R	O	C	
Acute gastritis 5	Sulam	...	...	...	2	2	...	...	...	4	...	1	3	
	Amasulam	...	...	...	1	...	...	...	...	1	...	...	1	
		...	...	...	...	...	...	...	...	5	...	...	...	
Subactute gastritis 1	Sulam	1	...	...	...	...	...	...	...	1	...	...	1	
Chronic gastritis 5	Vistabhdajirna	...	1	...	...	...	...	...	...	1	1		...	
	Vidaghajirna	...	1	...	...	...	...	...	...	1		1	...	
	Udavartha	1	...	...	...	...	...	...	...	1	1		...	
	Sulam	...	...	...	...	...	2	...	...	5		2	...	
Gastric ulcer 33	Soolam	6	2	3	1	6	1	...	1	20	14	6	...	
	Parinamasula	...	...	...	...	2	...	...	...	2	1	1	...	
	Gulma	7	...	...	2	2	...	...	...	11 33	7	...	4	

Pyloric ulcer 5	Parinamo sulam	...	...	...	...	1	...	...	...	1	1	...	...	
	Sulam	...	...	...	...	4	...	...	...	5	4	...	...	
Pyloro Deuodenal ulcer 1	Sulam	...	...	...	...	1	...	...	...	1 — 1	1	...	...	
Dueodenal ulcer 50	Sulam	5	1	...	4	5	...	1	...	16	15	1	...	
	Parinama sulam	9	5	...	1	16	...	1	...	32	25	7	...	
	Gulma	1	...	...	1	...	...	...	...	2 50	2	...	...	
				Grand total					...	100				

V = Vata
P = Pitta
K = Kapha.

R = Relieved
O = Otherwise discharged
C = Cured.

The cases that are put under 'R' are cases where all the signs and symptoms for which they were admitted, had completely disappeared. They were given normal diet and when pain had not occurred for a period of more than two weeks and had put on body weight they were discharged.

should be given. In the hospital though many of the diets that are prescribed in the books could not be given due to administrative difficulties a few diets as mentioned in the books are given. People who are accustomed to non-vegeterian diet, Dasa mula mamsa rasa (mutton soup boiled with Dasamula) is given. For vegeterians where milk is agreeable milk boiled with Dasamula or Lasuna gives very good results. In other cases kshirapayas (milk conjee) with rice or wheat gives good results. When the pain, vomiting and such other aggressive symptoms have abated solid food is given.

I acknowledge with thanks the help rendered by some apprentice physicians in collecting the various statistical datas.

Liberty Press, Madras.

Issue No. 3.

पुस्तकालय
गुरुकुल कांगड़ी

TRANSACTIONS
OF
THE ACADEMY OF INDIAN MEDICINE
MADRAS
1941-42

WESTERN VIEWS ON "ABDOMINAL PAIN IN RELATION TO FOOD"

BY

Dr. Khan Sahib SYED TAJUDIN BAHADUR

FOUNDATION FELLOW, THE ACADEMY OF INDIAN MEDICINE

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MODERN VIEWS ON "ABDOMINAL PAIN IN RELATION TO FOOD"

At the very outset I should very much like to express my thankfulness to the office-bearers of the Academy of Indian Medicine for having given me this opportunity for placing before the members of the Academy, western or allopathic views on "abdominal pain in relation to food." In the last meeting we already had the Ayurvedic aspect of the Subject.

Though the theme seems to be limited and circumscribed in its outlook, I am afraid, I will have at least to wade through almost all the ailments connected with the alimentary tract in order to provide a comprehensive view of the Subject. Since we have to traverse over a vast region within a very limited time, we will have to perform an aeroplane journey, as it were, which, at best would probably furnish us with but a bird's-eye view of the same.

Medical Synthesis, I believe, is or should be one of the prime aims of our Academy. As such we should endeavour to consider views according to different systems of medicine, prevailing in this vast land of ours, upon any and every subject that is discussed within its portals. Next time, I hope some eminent Hakim, from among the fellows, will provide us with the Unani aspect.

Gastric disease is probably as old as humanity itself. There seems to be much truth in the old adage—"the platter often kills more than the sword." That the digestive disorders are very wide-spread in India, nay all the world over, mainly due to gross ignorance and wilful indiscretions is well known to every practitioner of medicine. But it is not sufficiently recognised that intelligent treatment of these ailments must be broad-based on physiological knowledge. Without such knowledge the so-called treatment can only be empirical and it will prove at best but 'a shot in the dark.' But even though, as the result of experimental investigation, knowledge of gastric function and mechanism has considerably advanced within the last three or four decades and even though a good amount of light has been shed on several difficult and obscure problems of digestion, the knowledge of the digestive system and the knowledge of the human body in general is still somewhat rudimentary. It seems to be well-nigh impossible to grasp its constitution at present in its entirity. This statement might sound strange but the fact remains.

If modern physiology or modern medical science had fully unravelled the inner machinery of the animal or human body, it would or could have produced the same wonders as the Durvishes and the Siddhas or the adepts of Indian Medicine have worked. A hatayogi or a Rafai Durvesh (a sufi who practises Islamic hatayoga) is a better physiologist and scientist in so far as he is better acquainted with the inner and hidden mechanism of the human body. He can bring, for instance, the action of heart and lungs to a stand-still. He can practise 'Suspended animation' whenever he wants and likes. He knows the secret of producing suspension of nutrition or metabolism. He knows to control and hold sway over the involuntary muscles of the body. To him the modern physiological division of muscles into voluntary and involuntary, has no meaning at all. Such a knowledge may be called the higher physiology or super science of the human body. Where are those Siddhas and Durvishes who practise such a super science, the science of all sciences? I believe they still "live and move and have their being" in this scared land of ours! Of course such a knowledge is beyond the reach of 'Tom, Dick and Harry,' the vulgar herd or for the matter of that, the common run of Practitioners of Indian Systems of medicine.

Lord Curzon of Kedleston has recorded an instance in his travels in Africa of having personally witnessed Durvishes inflicting deep wounds on their own bodies with sharp weapons, which never bled but which healed instantaneously and on the spot! And pain was unknown to them. A few years ago, one by name 'Sarvabatchini' who was roaming all over the country, could eat any thing and everything with impunity (really omnivorus). Any rank or deadly poison he could take in the presence of thousands of people. When he, once, visited Tanjore I poured few ounces of fuming nitric acid in the hollow of his hand; he licked it up with zest with the tip of his tongue. I offered him a big piece of black aconite which he munched and swallowed. So he cast away to the winds all the allopathic ideas of Toxicology! Modern medical science has not discovered as yet these secrets. The knowledge of the human body is still meagre and superficial.

The oriental secret Science of 'Suspended animation' provides higher opportunities to exceptionally gifted individuals by enabling them to hibernate time after time. When some animals can hibernate to their advantage why not man? The elixir of life—Kayakalpam--seems to be not unknown to them. Not a mere myth of the dead past! I beg to be excused for this digression.

A working knowledge of the general anatomy and Physiology of human alimentary tract is imperative for the intelligent study of the digestive diseases. As far as the anatomy of the stomach is concerned, the matter is comparatively easy and simple. But when we come to consider gastric Physiology, the matter becomes very difficult. "We know" says Maclean "that certain Phenomena take place during the digestion of food but how the various functions are initiated and controlled is, in the comparatively meagre light of our present knowledge, frequently but a matter of speculation and conjecture"

The Structure of the Stomach :—

The stomach may be called a muscular bag lined by a mucus membrane studded with large numbers of secreting glands. It may not be wrong to regard it as a particular dilatation of the esophagus. The wall of the stomach is made up of three layers, longitudinal, circular and oblique. The distal or pyloric portion of the stomach is formed of a strong, powerful, and well-developed wall of muscular fibres. The mucus membrane which lines the stomach contains two different varieties of glands. These are known as pyloric and fundic, according to their situation. The pyloric region of the stomach contains pyloric glands. The fundic glands are the most numerous and occupy the whole body of the stomach excepting the cardiac and pyloric regions. One important point has to be remembered with regard to these gastric glands. In the pyloric region and a small area in the neighbourhood of the cardiac part of the stomach no acid producing glands are found.

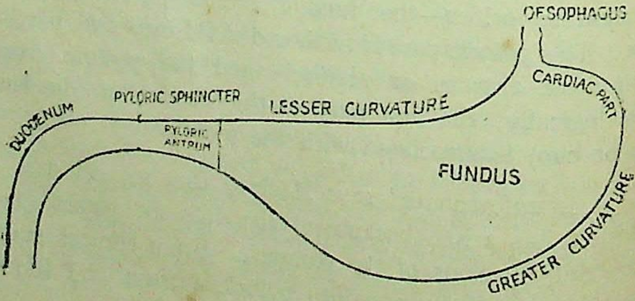
Clinical Sub-divisions :—

Human Stomach is of a simple type and it is not so complicated as that of many animals. Even the so-called simple stomach is from a physiological stand-point very complicated, for its different portions are not only different histologically but also perform specific functions. The human stomach is divided into two chief parts, the fundus and the pyloric antrum. The upper border is called the lesser curvature. The Pyloric antrum forms comparatively a small part of the stomach before it joins the duodenum. At the point of meeting of the stomach with the duodenum a thick circular muscle forms a sphincter which can shut off completely the duodenal lumen from that of the stomach. The pyloric sphincter plays an important role in the physiology of digestion.

The rest of the body of the stomach is known as the fundus. The fundus is at times sub-divided into a small part lying round the opening of the esophagus—the cardiac part—and the part between this and the pyloric antrum—the prepyloric region. Wright calls the part of the stomach to the left of the incisura angularis, the cardiac portion, and to the right, the pyloric part. The cardiac portion is divided into a part above the line of the cardiac orifice—the fundus and a part below—the body. The pyloric part is further divided into two parts—the pyloric antrum or vestibule and the pyloric canal. Physiologically, the first part of the duodenum (duodenal bulb or cap) is associated with the pylorus.

'It is unfortunate' says Maclean that a good deal of confusion has arisen because of the different names given to the Sub-divisions of the stomach. From clinical standpoint the general division into pyloric antrum and fundus is very convenient for these two portions of the stomach differ markedly in anatomical structure and physiological activity."

Hurst and Stewart say that the term pyloric antrum should be discarded, as it has been employed with so many meanings that its retention can only lead to confusion. They divide the stomach into a larger cardiac part and a smaller pyloric part. The cardiac part consists of the fundus and body, the fundus being the segment of the stomach which lies above a horizontal plane passing through the cardiac orifice. The body of the stomach is situated entirely to the left of the middle line. The division of the stomach into two parts is physiological rather than anatomical. The pyloric part of the stomach consists of the pyloric vestibule and pyloric canal. The pyloric canal is about half or three-quarters of an inch in length which is normally closed by the tonic action of the pyloric sphincter. The cardiac sphincter which is formed with the circular muscle layer of the oesophagus, at its entrance into the cardiac end of the stomach, shuts off the stomach cavity from the oesophagus during digestion.



Position of the Stomach :—

The stomach is not a fixed organ. Its position varies not only with the position of the body but also with the amount of food or gas present. When the body is in the horizontal position, the stomach is found in an oblique transverse position from left to right in the abdomen When it is empty its walls are fairly in opposition.

Movements of the Stomach :—

The preparation of food for further treatment in the intestine by the pancreatic and intestinal juices is its most important role. The motor activity and digestion with the aid of the stomach enzymes are the two factors that participate in the preparation of food. In the mouth and in the stomach there is actual breaking down of food into a pulpy mass. The removal of the stomach is not incompatible with life provided the food is properly prepared beforehand and given in small quantities. But life would be impossible in the absence of intestinal digestion. By this it should not be meant that the stomach is the fifth wheel of the coach.

Relation of Stomach movements to Nerves :—

The intrinsic nerve supply to the stomach is provided from a double source—the vagi and the sympathetic. The posterior surface is supplied with the right vagus and the anterior surface with the left vagus. The muscular tissue and the mucus membrane contain an intrinsic nerve plexus. The motor nerve of the stomach is the vagus which also probably controlls the pyloric sphincter. The sympathetic is antagonistic to the vagus in its action and acts as the inhibitory nerve of the stomach. External causes affecting the central nervous system may influence both the motor and secretory activity through these nerves. Since various nervous and toxic states are often associated with gastric disturbances, a general well-being of the patient is very important. Canon's experiments on digestion in a cat revealed the interesting fact, that its stomach movements immediately ceased as soon as the animal got excited or enraged. Like the heart, the stomach is to a less or more extent automatic since its activity is not entirely dependent on intrinsic nerves. Even when its external nerve supply is completely cut off, it can function, both as regards movements and secretion. Extrinsic nerve impulses are necessary normally for the regulation and control of its functions to the best advantage. 'In this respect' says Maclean "the stomach may be compared to a race horse which though quite capable of independent action is not likely to run a race except under the regulating and guiding influences of its rider."

The physiology of digestion and its bearing on digestive diseases :—

Food undergoes chemical disintegration in the alimentary tract through the influence of various enzymes, which have particular action on particular types of food. In this way the particles of food are reduced to minute fractions which get absorbed into the system through the blood and provide heat and energy. Before protein is built up in the body, partly built substance—premature protein—should be provided. 'In this respect' says Maclean "organisms resemble a bricklayer who can build houses when bricks are provided but who is himself unable to make bricks." Amino-acids are the simplest bricks that go to form the protein edifice.

All starches are broken down and reduced into sugar. These enter the body as glucose. Fats are broken down into fatty acids and glycerine. There are several stages in the splitting up of protein into amino acids and the process of disintegration is complicated. The stages of conversion are as follows.

Protein—> Proteoses—> peptones—> Polypeptides —>aminoacids.

Starch—> Starch—> erythrodextrin—> Achrodextrin —> maltose—> glucose. The splitting of *fat* is simple. The fatty acids are separated from the glycerine.

What happens when a morsel of food is taken in the mouth ?—

A reflex action is set up and a copious secretion of saliva takes place. Ptyalin is the enzyme of saliva which breaks down the starch into sugar, acting very rapidly. If any starchy food, such as rice or potato is well-masticated and examined at once for sugar, by the usual Fehling's test, a definite sugar reaction can be observed. Alkaline medium is the best for the action of ptyalin. Weak alkaline one is not so good. Acid medium is very detrimental. Even such a low concentration of 0-004% of acid would kill it instantaneously. When starchy foods are taken, good amount of sugar is produced in the alimentary canal.

Pepsin is the only important enzyme of the stomach. Its function is to disintegrate proteins. For its action, the presence of hydrochloric acid in the stomach is necessary It is not such a powerful hydrolyser of protein. Hcl is produced by half moon-shaped cells found in the fundus. There are various views about the exact mode of formation of the acid.

Gastric digestion is not considered as an essential process. It simply prepares the proteins for the stronger action of pancreatic and intestinal enzymes. Rennin, another enzyme of the stomach coagulates milk. The percentage of hydrochloric acid is from 0·4 to 0·5 in pure gastric juice unmixed with food. During the height of digestion, the actual percentage is about ·15 to 0·2 or a little more. Gastric juice is secreted during the initial stages of digestion. According to Haneborg psychic stimuli are most responsible for more than one fifth of the total gastric secretion. Further gastric secretion is induced by the presence of food in the stomach. Certain kinds of food excite the secretion more than others. Meat extracts and meat juices, for instance, are exceedingly active agents. Milk, bread and white of egg are not so effective; slight secretion is caused by water. Meat juices and extracts would only render the patients with hypersecretion worse.

The intestinal juices :—

By far the most important digestive processes occur in the intestine. When food leaves the stomach and enters the duodenum, the intestinal digestion begins. The whole process is over by the time the products reach the large intestine. The Pancreatic juice, the bile and the succus entericus (Intestinal juice)—help digestion in the intestines. Of these the pancreatic is the most powerful. All classes of food stuffs are dealt with by the pancreatic enzymes, namely (1) Trypsin—(protein splitting) (2) Amylase (starch splitting) and (3) Lipase (fat splitting).

Trypsin :—

When peptic digestion stops with the production of peptone, trypsin acts on the peptone and splits it into its ultimate compounds—the amino acids. Trypsin does not require the aid of pepsin for the hydrolysing. Cases in which active pepsin is absent in the stomach, do not suffer from any symptons, as all the necessary changes are carried out by trypsin which acts best in alkaline solution of fair strength. It is, however, capable of hydrolysing protein in neutral or even mild acid solutions.

Amylase is a converter of starch just like ptyalin. It is a stong enzyme. If starch escapes digestion by saliva, this completes the hydrolysis. Most of the starchy food, if not all, is completely converted into sugar in the upper portion of the intestine. Maltose is the sugar that is actually formed while maltase of the intestine changes it into glucose.

Lipase is also an important enzyme. It splits fat into glycerine and fatty acids. Fat gets emulsified. "Fat is absorbed as free fatty acids and glycerol and is resynthesized after absorption" (Maclean). It is strange that when bile is obstructed from reaching the intestinal canal by impacted-stones, fat seems to be hydrolized quite well.

Pancreatic secretion commences a few minutes after the beginning of a meal. It increases rapidly when food enters the duodenum and continues for 2 or 3 hours. It is controlled by the vagus and by a chemical mechanism—'Secretin' a hormone formed in the duodenum. It neutralizes approximately equal volume of gastric juice.

The chief Gastric diseases.

Gastric diseases are divided into two main classes—functional and organic—depending on the absence or presence of some definite lesion in the stomach. The classification of the functional disorders is, indeed, difficult

In most of the text books, a statisfactory grouping is not found. So far, Sir Robert Hutchinson's classifications seems to be the best. He has grouped them under three main heads :-(1) Physiological (2) Chemical (3) Eteological.

1. Physiological classification :—

In this, cases are arranged according to the particular function affected.

(a) Affections of Secretion.

- i. excessive—Hyperchlorhydria, hypersecretion,
- ii. defective—Hypochlorhydria, subnormal secretion or Achylia gastrica.

(b) Affections of Motility.

- i. Excess—Pyloric or gastric spasm.
- ii. Defect—Atony or impaired Motility.

(c) Affections of Sensation.

Excess—Hyperaesthesia or gastralgia.

Any of these may be present alone or two or more may exist in conjunction. The diagnosis of the disorders of secretion and motility can easily be made with the aid of the stomach tube and by the x-ray—These are the two chief modern methods of making a diagnosis.

2. Clinical classification :—

Cases of functional dyspepsia may be classified into certain rough groups according to their symptoms.

(a) Hypersthenic or hypersecretion dyspepsia. Probably due to a combination of hypersecretion and hypermotility.

(b) Asthenic or Atonic. This is due to impaired motility (atony) with or without some disorder of secretion.

(c) Acid Dyspepsia. This is an ill-defined group in which the chief symptom is a sensation of acidity or the presence of sour belchings. Some cases are really examples of hypersecretion with or without the presence of gastritis. In others the cause is the production of organic acids by fermentation.

Definite diagnosis can only be made by the aid of stomach tube and analysis of a test meal.

3. Eteological classification:—

Instead of attempting to distinguish different forms of functional dyspepsia one can regard the latter as an aggregation of symptmos of gastric disorder excited by different causes and classify cases according to the particular exciting cause at work. This is simple and useful for purposes of treatment.

(a) Dietetic causes—*e.g.* unsuitable food, hasty meals, the abuse of alcohol, tabacco, tea etc.

(b) Physical causes—*e.g.* imperfect chewing, defective teeth, oral sepsis, overfatigue, deficient exercise etc.

(c) Mental causes—*e.g.* over work, a studious life.

(d) Emotional causes—*e.g.* shock, worry, love affairs etc., Probably a combination of the first and third methods which take into account both the particular disorder which is present and the cause which has brought it about will lead to the best treatment.

It is well known that a great deal of the suffering is associated with some functional disorders. In a large majority of cases we do not know the exact nature of such disturbances. We only hazard a conjecture.

"We call gastric ailments functional" says Maclean "when we can find no evidence of pathological lesion, when definite lesion is present, it passes into the category of 'organic disease.' To ascertain whether a patient is suffering from a functional or organic disturbance is of paramount importance and frequently constitutes one of the most difficult problems in the investigation of gastric disease." The Subject, therefore, bristles with riddles and enigmas. The history of gastric disease, during the last few centuries, is an interesting illustration of many an obscure question.

Occasionally, it may be very difficult or even almost impossible to say whether a patient is really suffering from an organic lesion but a thorough and systematic examination may very often reveal its true nature.

If we adopt Maclean's classification, the chief diseases are four in number, though many names have been applied to stomach disorders. Various publications provide us formidable lists of gastric ailments. The following conditions comprise all the diseases that are generally encountered in practice.

1. Functional disorders :—

This group is sub-divideded as *(a)* Hyperacidity *(b)* Hypo-acidity or sub-acidity *(c)* Dyspepsia of general debility *(d)* Nervous dyspepsia.

2. Gastritis }
3. Ulcer } These three form the main organic disorders.
4. Cancer }

When pressure elecits definite pain, one may be almost sure that some organic lesion is present. This is true in the great majority of cases. This is a most important point in differentiating between functional and organic disease. Whereas functional disorders are not associated with tenderness.

Discomfort and pain in upper abdomen :—

Most of the stomach ailments, whether organic or functional are associated with less or more degree of discomfort in the upper part of the abdomen. At times the discomfort may be only a mere sense of uneasiness which the patient hardly notices. In some other cases, severe pain may be felt. The painful symptom may range from a trifling inconvenience to steady incisive or cutting pain. Some patients feel the discomfort as if a ball of lead is present in the stomach. Sometimes the discomfort gets localised or circumscribed, but as a rule it spreads over almost the whole part of the region above the umbilicus. Pain, even when it is not felt otherwise, may be elicited on pressure.

Heart burn is the expression to describe a feeling of burning felt at the pit of the stomach. A similar burning sensation is felt also in the throat and the upper part of the sternum. This symptom is due to the presence of excessive hydrochloric acid, due to hypersecretion in the stomach, and at times, this symptom is due to the presence of organic acids such as lactic and acetic formed as the result of fermentation.

Functional Diseases.

The condition of the nervous mechanism of the stomach is of fundamental importance in the causation of functional gastric disease. Since various external stimuli govern this nervous mechanism, a patient's mental and emotional state plays an important role in this connection. Though exactly the very same symptoms may be presented by functional diseases as in organic disorders, there are certain conditions which are characteristic of functional ailments. The occasional co-existence of organic and functional disease in one and the same subject may complicate the problem.

With the present day steady and uniform advance in our knowledge, undoubtedly certain conditions, now looked upon as functional in character, will eventually be marked as organic in their very nature. All the same there are probably always a certain residual number of gastric disorders that justifiably may be regarded as primarily functional in their true nature.

Let us now take up the principal forms of functional gastric disease, one by one.

Hyperacidity.

This is by far the commonest form of functional disorder. Hypersecretion signifies an increase in the amount of gastric acid produced. The excessive acidity of the gastric juice is a potential danger as this condition is often a forerunner of gastric ulcer. It is a clinical syndrome in which there is both gastric secretion and epigastric distress. Some authors say that both must be present to warrant the designation hyperchlorhydria. The stomach possesses an automatic or self-regulating mechanism. When the gastric juice reaches a certain acid-concentration, further secretion of acid is stopped. Wonderful indeed! In hyperacidity for some unknown, though not, perhaps, for ever unknowable reason, this wonderful mechanism is more or less out of gear, with the result that stomach keeps on secreting long after the normal restraining concentration is reached. This is a simple way of explaining what hyperacidity means so far as our present knowledge goes.

Besides high acidity during gastric digestion, there is in a large majority of cases, a continuous secretion of gastric juice while the stomach is empty in the interval between meals. In hyperacidity either the actual amount of gastric juice may be increased or a juice containing a higher percentage of acid than normal may be secreted. It is generally held that if more than 30 c.c. of secretion can be obtained from the resting stomach, hypersecretion is present (Maclean).

'This is not' says Mclister 'a disease entity; it is a clinical syndrome in which there are both gastric hypersecretion and epigastric distress; both must be present to warrant the designation, hyperchlorhydria. Increased gastric acidity is not incompatible with health and comfort. It is believed to accompany the widest variety of abnormal states. Many authors state that this syndrome is a frequent accompaniment of nervous disturbances such as vagotonia and emotional imbalance. Others believe that it represents always some organic disease within the abdomen nearly always, if not always, such as peptic ulcer, appendicitis or cholecystitis. As a matter of fact, the condition may be due to a reflex influence from other abdominal diseases as those of gall bladder or appendix. No doubt it is frequently found associated with organic disease of the stomach (gastric or duodenal ulcer, and in the acid forms of chronic gastritis). In many cases, however, there is no organic cause and it may be of pure nervous origin.

It occurs in two forms (1) continuous (2) paroxysmal or intermittent.

The Symptoms which characterize hyperchlorhydria are pain at the cardiac orifice of the stomach, acid eructations (sour belching), water brash, usually flatulence and constipation. The patient very often can tell the exact hour when discomfort begins. It is generally one to two or two and a half hours after breakfast but the time varies in different cases or at times in the same case. The pain may fade away, lasting for an hour or two, only to reappear an hour or longer after the midday meal, partaking of this meal removes it for a time, the period of relief varying from half an hour to an hour or longer. The duration of the intervel of comfort depends on the severity of the condition and the quantity and the composition of the midday meal. This sequence of events,—meals, interval of relief, recurrence of pain or descomfort—may be maintained throughout the rest of the day. If the last meal of the day be taken near bed time, the patient may be awakened from sleep by pain and other symptoms which may keep him awake for an hour or two. The pain or discomfort varies within wide limits. They may be severe and acute and described as grinding, gnawing, boring, burning or the sensation may be dragging, of weariness, of distension, of a hunger discomfort. The pain goes through to the back near the spine; it may be present behind either shoulderblade or be most marked in the normal position of the pylorus or over the liver behind.

Wylle called this pain as 'delayed pain' to distinguish it from the pain that comes on in other stomach disorders immediately after or soon after taking food. The characteristic feature of pain is that it is nearly always relieved by the ingestion of food or even water, the taking of alkalies or by vomiting. The pain is induced more by starchy food than by proteins. A large protein meal at dinner time may not give rise to discomfort for over two hours or even three.

In the extreme cases of constitutional hyperchlorhydria the stomach secretes acid throughout the night, so that undiluted gastric juice comes into contact with the mucus membrane of the stomach and duodenal bulb for 9 or 10 hours. Moreover, owing to the deficient power of secreting mucus, which is characteristic of the hypersthenic stomach, the protection against damage afforded by a layer of mucus in the hyposthenic stomach is absent. Hyperchlorhydria is very frequently associated with minute erosions, some of which may develop into acute ulcers, which may in turn become chronic ulcers (Price).

Treatment :—

This condition by itself does not usually require treatment, but, when it is attended with symptoms of heartburn, acid eructations and pain after food, bland diet should be given. Food stuffs that stimulate gastric secretion should be avoided. Those articles of food which best 'fix' free hydrochloric acid or which tend to restrain its production should predominate in the diet.

Therefore vineger, salt, pepper mustard, spices, condiments, and pickles should be avoided. Fats, oils and animal foods should be taken freely but starch foods should be restricted. Warm milk between breakfast and lunch and in the afternoon instead of tea and at bed time should be had. Over-smoking and alcohol should be prohibited. The times of meal should be regulated. The interval between two meals or feeds should not be longer than 2 or 3 hours. Antacids, though useful, should not be persisted in. Carbonates of calcium, magnesium and bismuth are generally given. Tablets of 10 grs. may be given between meals. Sodabicarb and Aluminum hydroxide (Alocol) are also useful. Details of alkali treatment will be found under gastric ulcer. Tr. Belladonna (5 ms. doses) is at times given before meals to restrain secretion.

Hypochlorhydria, Achlorhydria and Achylia Gastrica :-

These represent different stages of a disorder of the secretory mechanism of the stomach characterised by the diminution or cessation of the secretion of hydrochloric acid. According to R. F. Soel (The Practitioner's Library of Medicine) complete absence of free acid is known as achlorhydria and when the ferments too are abolished achylia gastrica is said to exist which is the end stage in the progressive loss of gastric secretion. He says that subacidity of the gastric juice may exist in the presence of an apparently healthy mucosa or may result from an actual progressive degeneration of the acid bearing cells of the the stomach.

Two forms of achylia gastrica are recognized by J. Sailer (Tice, Practice of Medicine), the Secondary and the Primary. In the Secondary form the failure of the gastric secretion is associated with some other condition which may be regarded as the cause in the primary form; no adequate explanation can be found.

Achylia gastrica is probably the result of congenital changes and does not seem to arise as an acquired defect usually. The estimation of total inorganic chlorides will enable us to ascertain how many of the Achylia cases really secrete no gastric juice at all. The three chief pathological conditions associated with subacidity are (1) Gastric cancer (2) Pernicious anaemia (3) Gastritis.

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In achylia gastrica nocturnal pains similar to those observed in cases of duodenal ulcer rarely occur. The patient is usually disturbed from sleep about two in the morning. Local heat or food may allay the pains. But alkalies have no effect. The symptom of pain is relieved or abolished by the acid treatment. These are not associated with hunger pains. In achlorhydric gastritis, if uncomplicated, pain never occurs. Only slight epigastric discomfort is felt immediately after food and it may last for several hours. In these cases there may be a history of vague dyspepsia and uneasiness in the epigastric region with loss of appetite and occasional nausea. Hypochlorhydria is not uncommon in elderly people. The real treatment of subacidity is the removal of the cause; when this is not possible, the acid treatment gives only temporary relief.

Dyspepsia of general debility.

In this there is a marked lack of tone in the gastric musculature giving rise to atony and dilatation. The proper treatment should be tonic in its very nature.

Nervous Dyspepsia.

Sir William Osler says that this is not a satisfactory term but it is difficult to find a better one to designate the condition in a large group of patients who complain of gastric disturbance without any organic disease of the stomach or elsewhere in the abdomen, apparently secondary to neurasthenia and allied conditions. There is general fatigue, psychical, emotional and physical. The digestive symptoms are characteristic only in their irregularity and uncertainty. Discomfort is a common complaint usually worse after meals but varying in

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occurrence and severity. As a rule, it has no special relation to any particular food unless the patient gets the notion that some particular food is responsible and expects trouble. Then it comes as expected. Frequently symptoms fit in with any diagnosis so far as gastric ailments are concerned; that is why Nervous Dyspepsia is one of the bugbears of the clinician. Though many cases can be diagnosed with ease, it is sometimes exceedingly difficult to be certain whether or not an organic lesion is present and the most experienced doctors very often make mistakes. The condition may simulate in all particulars some other gastric disease such as ulcer and, therefore, the greatest care should be exercised in dealing with such cases. The symptoms generally vary from hour to hour and day to day without any apparent reason. Nervous Dyspepsia is very common in hysterical and neurasthenic subjects; these patients feel worse in the morning and better as the evening advances and improve by night. Nervous dyspepsia may be polysymptomatic or monosymptomatic. Physical and radiological examination of the stomach rarely, if ever, shows any abnormality. The digestive symptoms are always associated with other nervous symptoms such as headache, sleeplessness etc.

Treatment :—

The general neurasthenic condition requires attention and treatment first and foremost. The digestive condition may not require special treatment.

Gastritis.

'In the past' says the contributor to the British Encyclopedia of Medical Practice "the name Gastritis has been widely applied to disorders of gastric function without reference to exact symptomatology and pathology. At the beginning of the century the development of methods of precision in gastric diagnosis by means of improved operative methods, radiology and fractional test meals, leads to the better understanding of peptic ulcer and cancer, but gastritis eluded recognition and when a definite lesion could not be demonstrated, it came to be thought that gastric symptoms were of reflex or nervous origin and for a time, the term 'nervous dyspepsia' almost took the place of chronic gastritis. The pendulum is now swinging back as means of examining the inside of the stomach with the gastroscope during life has been evolved and methods of histological study of the tissues in an absolutely fresh state are being perfected " In the last 15 years there has been increased interest in gastritis as fresh gastric tissue is available for study, since resections for benign and malignant affections have been in vogue. It is an astonishingly common disease. It exists sometimes with and sometimes without symptoms. At times it is associated with peptic ulcer, cancer, anaemia or achlorhydria. The first International Congress of 1935 of gastroenterology has opened up a very wide field of investigation. However, many years should roll down before the chapter on gastritis is written in full. Eusterman offers the following clinical types of Gastritis :—*acute* gastritis, exogenous, corrosive and phlegmonous; *subacute* gastritis, usually secondary to obstructing lesions of stomach or duodenum or of infectious origin; *chronic* gastritis, primary, or secondary. From the standpoint of histology this division is based on the predominant type of gastritis present, as different degrees and stages of inflammatory changes are frequently found in the same specimen. So its nomenclature is complicated. Eusterman classifies gastritis in the year book of medicine on the basis of etiology, anatomic and functional changes, gastroscopic findings as well as clinical picture and course.

In gastritis there is always a tendency for defective gastric secretion and in well-marked cases there is little or no hydrochloric acid present in the gastric contents. No doubt gastritis in its earlier stages may be associated with a condition in which even excessive amounts of acid are secreted. (It is not due to influence of gastritis but to some other factor). It is really the excess of hydrochloric acid that sets up the gastritis and though hyperacidity and gastrits may exist together for some time, yet the more definitely the gastritis becomes established, the less acid is secreted until ultimately no acid at all will be found. Gastritis, *per se*, cannot increase the amount of acid and there is really no such condition as acid gastritis.

Acute catarrhal gastritis.

In this there is severe irritation of the mucus membrane of the stomach. In gastric influenza, scarlet fever, septecaemia and other acute infections, the condition of gastritis is produced by the toxins in which recovery takes place only after several days but in ordinary catarrhal case recovery generally occurs in 24 or 48 hours. In acute cases 'Acute abdomen' should not be overlooked.

In acute gastritis pain is the most common symptom. There is a feeling of weight or oppression at the epigastrium and in many cases actual pain which is increased by pressure or by the ingestion of food. It occurs most often in children in whom it is usually traceable to errors of diet. Acute gastric catarrh may be confounded with the early stages of enteric fever. Acute gastritis associated with infection is often unrecognized.

Phlegmonous gastritis.

Admittedly it is very difficult to diagnose this as the symptoms are so variable since the condition is very rare; the possibility of its occurence is most often, if not always, forgotten. Acute suppurative gastritis is caused by streptococci or pneumococci or staphalococci or B. coli of the submucus tissue through a cancerous ulcer etc. In Mycotic and parasitic gastritis, Fungi usually grow in the stomach and exite inflammation.

Toxic Gastritis.

This intense form is caused by swallowing concentrated mineral acids and strong alkalies or by such rank poisons as corrosive sublimate—mercuric chloride, arsenic etc. In the non-corrossive poisons, the deleterious process consists of an acute degeneration of the glandular elements and haemorrhage. Powerful poisons extensively destroy the mucus membrane. Inspection of the mouth and pharynx, in many instances, renders the diagnosis usually easy. The examination of the vomit will signify the nature of the poison. The ordinary febrile gastritis is easily recognised. But the acute febrile form is so much alike to the initial symptoms of some infectious diseases that it is impossible in a day or two to arrive at a correct diagnosis.

Treatment :—If there is fever, rest in bed and hot applications to the abdomen should be prescibed. Encourage vomiting by giving freely a warm solution of sodabicarb (1 dr. to $\frac{1}{2}$ pint). If there is much pain morphia injection should be given. If it is the result of food poisoning castor oil should be given. During the acute phase nothing should be given by the mouth except sips of water. During convalescence, the diet and medicinal treatment should be the same as in chronic gastritis. Remove the original cause, if necessary by lavage also.

Chronic Gastritis.

This is a condition of disturbed digestion, associated with increased mucus formation, qualitative or quantitative changes in the gastric juice and with alterations in the mucosa. The term Chronic gastritis has been used loosely to designate a variety of gastric disorders, in some of which there are no actual changes in the mucus membrane. It is a name frequently given to longstanding cases of Dyspepsia. Gastritis in the strict sense of the term implies a condition in which a definite inflammatory lesion in the mucus membrane of the stomach ultimately ending in atrophic changes.

The most characteristic feature of this disease is the presence of an excessive amount of mucus in the stomach contents. Vomiting especially in the morning occurs. Absence of free hydrochloric acid in the stomach may suggest the presence of cancer. If the stomach is washed and a test meal given, lactic acid is found when the contents are withdrawn after an hour in cancer cases. Oppression or feelings of distress after eating are among the early symptoms which may become aggravated and amount to actual pain. When the stomach is empty, the patient may also have a painful feeling. The pain differs in different cases and may be trifling or of extreme severity. "When localized and felt beneath the sternum or in the præcordial region" says Osler 'it is known as heart-burn.' There may be pain on pressure over the stomach, usually diffused and not severe. In the chronic catarrh of old topers a bout of morning vomiting is common in which a slimy mucus is brought up. In the so-called "acid gastritis" there may be increased hydrochloric acid.

Gastric Contents :—

The fasting stomach usually contain much mucus. Pathological organisms are almost constantly found of which streptococci are the most important. The hydrochloric acid is usually greatly diminished or absent. It may be found after stimulation by histamine which is the most potent excitant of acid gastric secretion known. The histamine test is the most rigorous test that may be employed to differentiate between true and false anacidity. In advanced cases with atrophy of the mucus membrane, mucus is absent and there may be neither acids nor ferments.

If any patient goes to you with Gastric Symptoms you should first decide whether he has any primary organic disease of the stomach or his condition is secondary to disease elsewhere. Gastritis is a common sequel of the congestion caused by the diseases of the heart, lungs and the liver. It is very often easy to fix one's attention on the area of symptoms and fail to recognize the site of the cause. Excluding cancer, especially in its early stage, which may give very great difficulty, the problem then is to decide whether there is any other organic change in the stomach such as ulcer or dilatation or whether the symptoms are purely functional.

Treatment :—

As regards treatment, the essential point is to discover the cause which, if possible, should be removed by proper measures. In many cases, the symptoms are secondary to disease elsewhere and in such circumstances, the treatment is largely of the primary condition.

A rigid milk diet may be tried. "Milk and sweet sound blood differ in nothing but colour" (George Cheyne).

The diet "says Sir Robert Hutchinson" should be planned to avoid all mechanical, chemical or thermal irritation of the Gastric mucus membrane. Woody and stringy vegetables, spices, condiments, fried fats, cane sugar, too hot and too cold foods should, therefore be avoided. Oral sepsis should be dealt with and the use of alcohol and tobacco should be forbidden. The abdomen should be protected by a warm belt. If there is much mucus in the vomit, lavage of the stomach before breakfast with a weak solution of of Soda-bicarb, or of hydrogen peroxide (1 to 2 drs. to the pint will be useful. Removal of the cause, if it is direct, or treatment of the underlying disorder, if it is indirect, should always be remembered. The prominent indirect causes, among others, are tuberculosis, syphilis, cirrhosis of the liver, nephritis, diabetes, myocardial failure and polyarthritis.

Gastric and Duodenal ulcers.

Both these conditions have much in common but there are many distinctive features. The eteology of gastric and duodenal ulcers is still unknown. A world of speculation and a most voluminous literature have not yet been able to clear up the mystery. One of the best discussions on ulcer is to be found in Boulton's 'Ulcer of the stomach.' One significant fact, however, does stand out prominently amid the chaos of speculation. Ulcer is always associated with the presence of hydrochloric acid. Ulceration is found only in the regions in which hydrochloric acid occurs. The "hydrochloric area" as it might be called, comprises the stomach and the first part of the duodenum. It is in the pyloric and adjacent duodenal region that prolonged and intense contact with the hydrochloric acid takes place. It is in this situation that ulceration is most frequent.

In regions far removed from the pylorus, ulceration is comparatively rare. As said before, one important factor in the causation of ulcer is excess of hydrochloric acid. Its deleterious action must be preceded by some local injury or lack of vitality in the particular area that is eventually to form the ulcer. It is the nature and cause of this injury that is puzzling. Bacterial infection is frequently blamed. Such an infection may produce its effects either by means of some toxin which is presumably eleminated in the ulcer area or by local infection, causing an inflammatory reaction or a small abscess. Probably there seems to be a kind of diathesis or ulcer constitution, which under favourable conditions predisposes to ulcer, for many members of the same family suffer from such ulcer (ulcer families). Occasionally operations have revealed an active ulcer in the duodenum and another in the stomach one and the same time. There is little doubt that woody vegetables and articles like nuts, tough skins of fruits and other indigestible foodstuffs frequently play a part in the eteology. A similar ill-effect is produced by a high protein diet which excites an abnormal flow of gastric juice. According to Sir William Osler, there are many suggested eteological factors, namely disturbance of the circulation (embolism, thrombosis or spasm of the artery) neurogenic influence acting through the sympathetic system (influence of a centre), the action of the acid, inflammation and the effect of general nervous strain and exhaustion. Probably various causes operate in different patients.

Gastric Ulcer.

Herein the usual symptoms of dyspepsia occur. The most marked feature is epigastric pain. A patient having a symptom-producing-ulcer, usually feels quite well in the morning before breakfast but the pain is post-prandial. The pain of gastric ulcer has several characteristic points depending on **the position of the ulcer**. If it is near the cardiac end of the stomach, pain comes on almost immediately after food. In typical cases pain gets worse and worse until vomiting takes place. After this the patient generally has relief. If the ulcer is nearer the pylorus, the on-set of pain is delayed for half an hour to an hour or more after partaking of food. As a general rule it may be said that nearer the ulcer to the pylorus the longer is the interval after food, before pain starts. Also in ulcers situated near the pyloric region, there does not seem to be the same tendency for the pain to cause vomiting, as is the case when the ulcer is situated near the cardiac end. The pain after reaching a climax gradually dies away leaving a feeling of discomfort and uneasiness behind. The intensity of the pain depends to a large extent on the nature of the food taken. The pain is much worse after solid than after liquid food.

In **pyloric ulcer,** the pain may not commence until three hours or even four after food and, unlike the typical gastric ulcer, is relieved rather than increased by taking food. In some cases of pyloric ulcer, food relieves the pain in exactly the same way as it does in duodenal ulcer.

The pain of **gastric ulcer** is always intimately related to food though the patient may complain of some sort of constant pain. Though continuous, this pain becomes acute at certain hours after food. This pain is always relieved to a less or more extent by taking alkalies. In the epigastrium, a certain small area of tenderness is found in addition to the pain. This area may be situated to the right or to the left of the middle line above umbilicus, but its exact position varies somewhat.

At times ulcer may be latent and only found post-mortem. In rare cases, according to Osler, it has no relation to food. In many cases it appears with the greatest regularity and punctuality as regards time after the same kind and quantity of meal so that “ the pain cycle ” reads as food, transcient relief, pain, comfort as contrasted with *the late and lasting pains of duodenal ulcer.*

If there is an association of gastroptocis (dislocation of the stomach) pain may be felt at the umbilical region. If the stomach is pushed or drawn to the right, the pain is felt further to the right than is usual. In a few cases it is felt high in the epgastrium, in some it has been substernal. It may even be referred well up in the præcordial region simulating pain of cardiac or præcordiac disease. Sometimes when the ulcer is on the posterior wall of the stomach nearer the lesser curvature, pain is referred to the chest or to the left of the spine, between the 8th dorsal and the second lumbar or about the 10th dorsal vertebera at which point there may be tenderness. According to Mayo, pain to the right of the middle line signifies duodenal ulcer and to the left gastric ulcer. The cases of continuous severe pain which is not relieved by taking food but only incompletely and temporarily by alkalies should be regarded to have caused a local peritonitis with adhesions to adjacent structures or by the resultant hour glass stomach.

According to some authorities, unyielding and unremiting pain may signify a transformation from a benign to a malignant condition at the site of a chronic ulcer, but it is rare.

Besides gastric ulcer, pain which refuses to yield to diet, rest, and alkalies is usually due to the perforating or indurative type.

The usual epigastric tenderness is explained by the increased sensitiveness of the solar plexus which is regularly observed in the presence of any active upper abdominal lesion.

Nature of the pain :—

In character it is intense, pressing, boring, burning, gnawing and even tearing. So pain is more severe than in any other gastric disorder. It is usually limited to a particular area. The patient will indicate the pain by placing his whole hand over the area whereas in duodenal ulcer he would point out with the tip of his finger. The pain is aggravated by food and relieved by vomiting. Seasonal incidence is not so pronounced as in duodenal ulcer.

In gastric ulcer, haemorrhage is present in about one-third of all cases. Occult blood may be found in the faeces. **Occult blood** simply means that the blood is so intimately mixed up with the faeces that its presence can only be detected by chemical means. Its presence practically always clinches the diagnosis. Hurst says that haemetemisis occurs in about 20 per cent of cases. An ulcer in the body of the stomach results in "hour glass" constriction (உடுக்கைப் போல்).

Duodenal Ulcer.

This occurs in the first portion of the duodenum. A chill is regarded as the most common exciting cause. Overwork and worry often bring on attacks; the symptoms are those of acid dyspepsia. The earliest symptom is generally a sense of discomfort or fullness about two or three hours after the largest meal. In its mildest form the pain may be nothing more than a slight sense of weight, pressure or distension. Vomiting is rare in uncomplicated cases when it occurs it is generally not entirely spontaneous. The patient vomits at will to get relief.

Sexual excitement invariably brings on pain in a patient of duodenal ulcer.

It is rather artificial to describe ulcers under the two usual headings of gastric and duodenal. As a matter of fact, ulcers in the duodenal region whether actually in the anatamical duodenum or in the stomach, present practically identical manifestations, whereas ulcers in the cardiac region are characterised by somewhat different signs. Symptoms intermediate between these two extremes or sometimes a combination of both kinds of symptoms are associated with an ulcer in the body, or prepyloric region of the stomach. 'Prepyloric ulcers,' says Hurst,' so closely simulate duodenal ulcers that it is generally impossible to distinguish them from the character of the symptoms. In duodenal ulcer there is marked flatulence, heart-burn and epigastric pain. This pain in its typical form differs from that of gastric ulcer in that it is relieved and not made worse by taking food ; this is one of the most characteristic points in the symptomatology of duodenal ulcer though a similar condition may exist in functional dyspepsia and in some cases of pyloric ulcer.

The pain of duodenal ulcer, generally starts two or three hours after food and is usually accompanied by a feeling of hunger. As a rule, very marked relief is obtained after taking food and the patient feels quite comfortable for some time after his last meal. The hunger pain relapses after some hours to be again relieved by food. In a typical case, the pain is much worse for two or three periods during the day and is absent in the intervals. When the patient goes to bed, he may feel quite well but gradually, as the stomach gets empty, the acid begins to irritate the ulcer and he wakes up with pain. This night pain is a frequent feature in duodenal ulcer. If the night pain has not been present in any form we may take it as fairly certain that it is not duodenal ulcer. Though intermittency of pain is a feature of duodenal ulcer, in some cases a certain amount of pain and discomfort is present all the time but the pain is always less marked after food; local tenderness is nearly always present but as in gastric ulcer, its exact site varies; it is generally found in the region immediately above the umbilicus but it may even be substernal. Though the tenderness may be absent in some cases, it may appear at some stage or other. On the whole, hyperacidity is a marked feature of duodenal ulcer and is probably always present at the earlier stages of the malady. There is little doubt that the general picture of duodenal ulcer varies in relation to the condition of the stomach in the vicinity of the lesion and elsewhere. The exact symptoms present, no doubt, depend to a great extent on the involvement of the mucus membrane by the irritative and inflammatory process present during acute exacerbation. Thus symptoms may on some occasions really appear to be those of acute gastritis. Indeed all sorts of combinations and permutations of symptoms may be observed in different cases of duodenal ulcer, so that it is comparatively rare to come across a case with the well-cut symptomatology of the text-books. One or two indications may be present and all the other manifestations may be quite anamalous. When a duodenal ulcer leads to pyloric obstruction the characteristic hunger pain gradually disappears.

Gross haemorrhage occurs in about 25 or 30 per cent of the cases.

Duodenal ulcers have no tendency whatever to turn malignant. Malignant growth in the duodenum is practically unknown (Maclean).

Carcinoma of the Stomach

The origin of the malignant tumour is as much a mystery as ever. It is a comparatively common disease, t any rate in the western countries, between the ages of 0 to 65, and is rare before thirty and after 65. Occasionally t has been obeserved in patients below thirty and ver 65. Indeed it may occur at any age, with the exception f perhaps extreme youth. It has been recorded in several atients under twenty years.

With regard to the eteology of cancer we know practically nothing. The danger of not recognizing a malignant rowth of the stomach at the early stage, when much can e done, probably by surgical treatment, is indeed very reat. In about seventy-five per cent of cases the symptoms ppear without any previous history of dyspepsia. In the emaining twenty-five per cent, there is more or less a airly long history of indigestion but some change in ymptoms is recently observable. The pain has become ess regular in its onset and comes on more often, directly fter meals instead of constant interval. The pain is ore severe and less easily and less completely relieved by ood, alkalies or vomiting and it never disappears completely xcept with rest in bed and careful dieting (Hurst).

If an individual, who has had hitherto no digestive rouble, suddenly begins to suffer from gastric symptoms he possibility of cancer should always be remembered and, f there is the slightest suspicion of cancer a fractional est meal should be given immediately. Absence of ydrochloric acid and the presence of notable quantities f **lactic acid** (more than 0·1 per cent) in test meal is sually conclusive evidence of cancer. The importance of he presence of lactic acid should be sufficiently appreciated by the medical profession. Though lactic acid may be absent even in cases of subacidity, its presence in appreciable amount is considered as a very bad omen and at times reveals the diagnosis even in the absence of symptoms. Hydrochloric acid is usually absent in chronic gastritis, pernicious anaemia and certain cases of dyspepsia, but in these conditions no lactic acid is present or if present at all only in very insignificant quantity.

It is stated in some text-books that a chronic gastric ulcer undergoes malignant changes and is followed by cancer. "How such statements ever came to be made is a matter for wonder," says Maclean "for careful examination of cancer patients," he continues to say "shows definitely that this story is but a figment of imagination. This is one other instance in which theory is shattered by fact." In Maclean's opinion, the 'ulcer cancer' bogy is one of the mysteries of modern mysteries. Records of hospitals conclusively show that cancer is not usually associated with previous dyspepsia of fairly long duration. Cancer, as usually met with is a disease of fairly sudden onset.

One should remember that dyspepsia and even chronic ulcer might be present in a patient who subsequently develops cancer without these conditions playing any part in the etiology of the cancer. Indeed from the prevalance of both cancer and ulcer it is certain that, on the law of averages alone, both cancer and ulcer must occasionally arise in the same patient and yet the one has nothing whatever to do with the other (Maclean).

In cancer there is an average previous history of symptoms of six and a half months but in ulcer the average period may extend over several years, say over seven years. It does not follow, however, that gastric ulcer never results in cancer at all. In a very few cases this happens. In these, ulcer plays but a trifling part in the eteology of cancer. Boas, who has had a vast experience in gastric pathology came to the conclusion that only three per cent of cancer patients exhibited any proof of previous gastric ulcer.

In benign ulcers, according to British Encyclopaedia of Medical practice, from 70 to 80 per cent of cases occur in the middle two-thirds of the stomach; from 10 to 20 per cent in the prepyloric region; in cancer of the stomach from 10 to 20 per cent are in the middle two-thirds of the stomach and from 60 to 70 per cent in the prepyloric region. These figures are decidedly against any close relationship between ulcer and cancer.

Occult blood is usually present in the faeces in cancer unlike to what is so often found in ulcer. The occult blood persists in cancer in spite of treatment but in ulcer it usually disappears with treatment. As the case advances emaciation becomes very marked. This is the usual picture of cancer.

In exceptional cases, on the other hand, the patient may look quite fit and have good appetite even when the greater part of the stomach is invaded by the growth. Inevitable fatal termination generally overtakes the patient in a year to eighteen months after the attack. Rarely patients survive three to four years.

Diagnosis of Gastric Diseases

From the practical point of view the chief extra gastric conditions giving rise to digestive disturbance are :—

(a) Cholecystitis.
(b) Chronic appendicitis.
(c) Spastic colon.

In all these conditions dyspepsia is more eratic and less dependent on the absence or presence of food in the stomach than is the case in stomach lesions. In hepatic lesions careful consideration of the nature of the pain and its relation to food may solve the problem. One very important problem in such cases is to endevour to ascertain whether the pain goes away suddenly. If it does so, the lesion is almost certainly in the gall-bladder and gall stones are present. The relation of pain to meal is the most important feature.

A thorough investigation is all the more essential since chronic appendicitis or chronic cholecystitis or both may be associated with a chronic gastritis or more frequently, a chronic duodenal ulcer.

Appendix Dyspepsia :—

Chronic appendicitis may give rise to pain in the right iliac fossa or in the epigastrium. It is only when the latter occurs that the term "appendix dyspepsia" can be correctly applied. The pain of chronic appendicitis is generally much less regular than in ulcer, its onset having no definite relation to meals, though it is often aggravated directly food is taken, an interval of complete freedom after a meal being comparatively uncommon. The pain is generally situated in the middle of the epigastrium. It frequently radiates towards the right iliac fossa, in some cases, pain may occur from time to time in this region independently of the more common epigastric pain.

The following points may be of help :—

(1) The epigastric pain precedes the right iliac.
(2) The epigastric or umbilical pain is present in some 50 per cent of cases.
(3) The right iliac pain is present in every case.

In ulcer, pain or discomfort is nearly always present at some time during the night and if this feature is absent in any suspected case the presumption is that the ulcer is not present. When the pain is eratic and has little relation to meal the condition is usually of nervous origin or it may be due to one of the reflex causes. The presence or absence of local tenderness is of great significance. For practical purposes of differential diagnosis there are only four conditions to be considered in ordinary gastric work namely.

(1) Functional dyspepsia.
(2) Ulcer.
(3) Gastritis. } organic disorders.
(4) Cancer.

The presence of definite tenderness at once removes the case from the first category and we know that any one of the remaining three conditions is probable. Local tenderness is always present at some stage or other of cancer and is usually evident in gastritis. In ulcer also it is very frequently observed though it may not be present while the patient is undergoing treatment. In gastritis and cancer tenderness will often be more or less general, but in ulcer it is usually limited to a definite circumscribed area in the epigastrium (Maclean).

Diagnosis from reflex Dyspepsia :—

As the epigastric pain in reflex dyspepsia is actually produced in the stomach as a result of reflexes from the disclosed organ, it may be identical in character with that caused by gastric or duodenal ulcer. In the majority of cases, the time of onset, the exciting causes and the means of obtaining relief are sufficiently indicative to prevent confusion. Moreover the associated symptoms such as sickness and nausea and the local signs and symptoms of the primary disease help to make the diagnosis.

Colon Pain :—

Spasmodic contraction of the intestines (enterospasm) may be a cause of epegastric pain which may simulate gastric pain by being induced by the taking of food.

The following observations of Behan regarding abdominal pain are very interesting and valuable.

"Pain due to Functional processes :—

The relationship if any, to the ingestion of food :—

If there is any such relationship, examine the stomach and intestines for a gastric or duodenal ulcer or for adhesions or the intestine for a volvulus or obstruction in which case the pain, at first is periodic and paroxysmal, and later continuous and of an aching dragging character. If the pain is sudden and intense especially if it commences in the umbilical region and gradually becomes localised to the right side, examine for appendicitis. If the pain is continuous and increasing, it indicates that the local peritonitis is spreading. This is especially the case should there be a synchronous increase in the tenderness. Biliary diseases, as a rule, cause a pain which comes on about the first or third hour after eating at the time of the greatest intestinal activity.

Pain in Gastric Areas

When pain occurs in the gastric areas, it is necessary to inquire into the following ;—

(1) The character of the pain.
(2) The time and manner of its appearance.
(3) Its relation, if any, to the ingestion of food.
(4) The duration of pain.
(5) The previous attacks.

Character of gastric pain :—

Certain types of gastric lesions have characteristic pains for instance that of gastritis is burning, that of spasm of the pylorus is sharp and sudden, that of ulcer is very severe and is sharply circumscribed, that of perforation is sharp and agonising and quickly spreads from the site of its original location, that of acute dilatation is severe and constant and is accompanied by symptoms of collapse.

It should also be borne in mind that the severity of the pain has no proportionate relation to the gastric conditions but often as the pain caused by lesions in other organs depends upon the susceptibility of the nervous system and the sensitiveness of the pain receptive centres of the individual patient.

Time and manner of its appearance :—

Should the onset be sudden without any apparent exciting factor and without any previous history of pain, perforating ulcer or pyloric spasm is to be considered. Acute gastritis is associated with the vomiting of indigestible or fermentating substances. This generally tells the tale.

Relationship to the ingestion of food :—

If the pain comes on at a definite time after eating and is relieved by vomiting, gastric ulcer should be thought of. If it comes on immediately after eating, the cardiac end is affected. On the other hand if it does not appear for an hour or two after eating, ulcer of the pylorus is to be considered. If instead of coming on immediately, it makes its appearance one half to two hours after eating, carcinoma and hyperchlorhydria should be considered. In carcinoma, the coffee ground vomit is distinctive while in hyperchlorhydria, the excess of hydrochloric acid with the absence of blood is sufficient to make the diagnosis. In early cancer, pain and coffee ground vomit are absent. Hyperchlorhydria may occur from gall-bladder diseases, duodenal ulcer or may be purely functional.

Duration of pain :—

The duration of the pain in any of the gastric disorders is variable. It seems to be present more constantly and for a greater length of time in those disorders which have an organic basis for their production. In cancers the pain is steady and persistent while in hyperchlorhydria and pyloric spasm it is associated with the ingestion of food.

Previous attacks :—

A history of previous attacks of pain may be of value in that it often is confirmatory of the diagnosis which the present symptoms suggest.

Associated symptoms : —

Constipation is generally associated with gastric ulcer, hyperacidity and cancer.

Pain reflected and referred to gastric area :—

When pain is present in the gastric area it may not be gastric but of nervous origin (such as intercostal neuralgia etc.,) or it may be projected from the cord (*e.g.* locomotor ataxia, general paralysis etc.,) or it may be reflex from other organs as the uterus, kidney, intestine, appendix, gall-bladder, pancreas, heart or prostate. Hyperchlorhydria is the only secretory neurosis of the stomach which produces pain. This pain comes on one half to two hours after eating and lasts a few hours or until more food is taken, being particularly relieved by the ingestion of proteids and also by alkalies. It is much less after large meals especially those containing much meat and eggs and deficient in carbohydrate. The pain differs from that due to gastric ulcer in being diffused and extending over the entire abdomen. It is generally precedes by a burning sort of pain which seems to run up under the sternum to the throat and is accompanied by the regurgitation of acid tasting fluid. The pain may also radiate to the back, to the asulla and to the scapula.

The pain of gastric ulcer :—

The longer the interval between the time of ingestion and the appearance of the pain, the farther away from the cardia and the nearer to the pylorus is the ulcer. If it has occurred immediately after food, the ulcer is probably near the cardiac orifice or the lesser curvature; if two or three hours after, it will be at the pylorus and if about four hours after relief ensues on taking food, is probably in the duodenum.

I am afraid I have tried your patience by spending much time on the various aspects of diagnosis. I am convinced, just as you are, I believe, that diagnosis is the soul of treatment. Sir Robert Hutchinson the great clinician once very pertinently remarked that "the first part of treatment is diagnosis, the second, diagnosis and the third, diagnosis." What aim is to a marksman diagnosis is to a medical man indeed. Otherwise it will be only a 'shot in the dark.' Without diagnosis, treatment is, therefore, a ridiculous hazard.

Treatment of digestive disorders.

Let us now consider the last aspect, namely treatment about which Sir Robert Hutchinson said "Without it the most accurate diagnosis is but a sounding brass or a tinkling cymbal."

'The sucessful treatment of gastric diseases' says Maclean 'depends upon a recognition of the cause of the condition present and a knowledge of the physiological processes involved in digestion. Obviously, local treatment applied to the stomach can be of little service, if the dyspeptic symptoms are dependent on a lesion in some other organ or are due to general conditions. Reflex causes of idigestion such as gall-stones and appendicitis, must be treated in the appropriate manner. In functional disturbances, suitable medical treatment will often work wonders. In organic lesions such as ulcer, the same is true. The most brilliant results can be obtained in gastric and duodenal ulcer, provided the physiological basis on which the principles of the treatment rest, is understood and the treatment is carried out with the necessary thoroughness.

If this is disregarded, the unfortunate patient will have to drift from bad to worse. Eventually his life becomes a misery. Only when medical treatment fails, surgical intervention should be indicated, but surgical treatment should not be understood as a cure in itself at all times. The patient may be free from the symptoms for a few months but the disease may return and the patient may suffer as much as ever. Until medical treatment fails, no kind of operation should be resorted to. It is indeed a bad principle to advise patients suffering from gastric or duodenal ulcer to undergo an operation at the earliest hour possible, after the diagnosis is established. Very many such cases can derive as much (or more) benefit by medicines as by surgery. There was a time when it was thought that, in order to prevent duodenal ulcers undergoing malignant changes, they should be cut off at the arliest moment but now it is definitely known that a uodenal ulcer never becomes malignant. Malignant egeneration of gastric ulceration is very rare and that om practical standpoint, Maclean thinks it is hardly orth considering. Perforation and severe haemorrhage ay warrant an operation, but such catastrophies are ew and far between in ulcer, that has been properly and arefully treated. At the same time one should not forget hat surgery has dangers of its own.

ndications for seeking Surgical intervention :—

(1) Extensive ulceration of the stomach.
(2) Ulceration, unyielding to medical therapy.
(3) Pyloric obstruction with scarring.
(4) Ulceration with perforation.
(5) Severe haemorrhage.
(6) Gross mechanical deformity of the stomach—*viz.* hour glass stomach—or of duodenum.

I do not propose to deal with the surgical aspect of the treatment. It is more the realm of a specialist in Surgery.

Medical Treatment

It goes without saying that all diseases, gastric or otherwise, are not radically curable. Radical treatment is a method which removes the cause and thereby automatically provides permanent relief. Many a gastric disorder, whose etiology is unknown, admits of only symptomatic treatment which is admittedly the second best as it aims at removing or alleviating the urgent symptoms in the hope that nature in the end will assert itself and bring about a cure. Many of the drugs employed are merely empirical agents, whose success, however, has been clearly established by fairly long experience. We should not be carried away with the notion that modern medicine is cent per cent scientific. Medical science is always in the making. "A large number of our most valuable drugs" says Sir Robert Hutchinson "are employed empirically."

Alkalies

In the treatment of the majority of the gastric ailments, alkalies, are, by far the most useful group of drugs. But for these the clinician would be, more or less powerless in counteracting the Symptoms of gastric disease. The reason for this is easy to find. Nearly all the functional and a good number of the organic, gastric ailments are associated with an increase of hydrochloric acid in the stomach and the easiest and the simplest method to do away with this excess of acid is to neutralize it by alkali. Hydrochloric acid in the free state is a most powerful substance which can destroy or disintegrate whatever it comes in contact with. Of course this property is useful in breaking down certain food stuffs but often the stomach itself falls a victim to the ravages of this disintegrating product. It cuts both ways.

When any abnormal change in the mechanism controlling its secretion results in an undue production of acid, even in the absence of food from the stomach, then this free acid gives rise to various symptoms. As the neuromuscular mechanism gets out of gear, a vicious circle is formed, the patient goes from bad to worse and much pain and discomfort result.

For combating acidity, the cheapest and the most effective alkali is soda bicarb. This is found naturally in the pancreatic juice. It gives immediate relief to the pain in most cases of ulcer. The quick relief is due in art to the immediate neutralization of the acid present the stomach and partly to the filling of the stomach with gass on the mechanism of the pyloric sphincter. However, it has one great disadvantage when it is present in xcess, it irritates the stomach and stimulates unwanted urther secretion of gastric juice. Hurst is also of the ame opinion as Maclean. If one can prescribe just the xact amount necessary to neutralize the excess acid in very case it would prove an ideal medicament but nfortunately this is not possible. To be effective, excess mount has to be given and this extra dose is the cause f the trouble. But the only way to obviate this difficulty s to combine some insoluble base along with the soda bicarb, so that if any more acid is secreted after all the soda bicarb is neutralized, the insoluble base will deal with it.

'The best all round mixture' says Maclean 'for gastric disturbances is one containing magnesia and calcium as well as soda. Whatever theoretical objections may be offered to this mixture—and very many of these objections to certain alkali are based entirely on theory—the following powder will be found to give excellent results.

R/	Sod. bicarb.	ʒss
	Heavy Mag. carb.	ʒi
	Calci carb.	ʒi
	Bismuth oxy. carb.	ʒii

ʒ i is a suitable dose but almost any dose may be given but it must depend on the severity of symptoms. It is best taken in suspension in soda water, or plain water or in a little milk. See that a sufficient dose is given.

Mag. oxide is regarded as another useful alkali. Magnesia cream is reputed to be a good preparation of magnesia.

It contains 24 grs. of suspended mag. hydroxide to the ounce. Its average dose is 2 to 4 drs. which can be given for long periods with impunity.

The proportion of magnesia and calcium in the Maclean powder should be varied according as the patient tends to get constipation or diarrhoea. The former tends to looseness and the latter to costiveness. Mag. carb. like soda bicarb. forms co_2 gas and so helps gastritis in dissolving muçus. Bismuth has only slight neutralizing effect ; sod. citrate prevents excessive curdling of milk and is a powerful antacid but at times it causes nausea. In milk feeds it is generally added in doses of 1 gr. for every ounce. Kaolin and chalk are also prescribed.

The antacid properties of hydrated Trisilicate of Magnesium are of particular value in gastric hyperacidity and chronic peptic ulceration. It has also strong antiseptic powers for protection of the ulcer base against digestion. It can be given in large doses without upsetting the general motility of the digestive tract, because it is completely insoluble in water ; any excess is voided in the stools. Mutch recommends the use of hydrated magnesium Trisilicate which has some advantage over the other antacids. It is a tasteless powder. It has more absorbent powers than bismuth, magnesia etc., and its neutralising effect is continuous for some hours after administration. The dose is up to sixty grains given four or five times a day. In larger doses it tends to looseness of the bowels. Krisner and Palmer think that of the various antacids studied, Calci-Carb in doses of 2 to 4 g. hourly, was the most effective neutralizer of gastric acidity. Stergmann and Fantus, after investigating the antacid properties of sippy powder No. 1 (Calci. carb. 1 part Sod. bicarb. 3 parts,) colloidal Aluminium hydroxide and a neutralyzed karaya gum, say that these agents give Symptomatic relief in a large percentage of cases without producing a uniform and constant reduction in the gastric acidity. Emery and Rutherford tried Aluminium hydroxide on some patients of peptic ulcer and concluded that in view of the severity of the cases, the results were very satisfactory. Mcintosh and Southerland employed colloidal aluminium hydroxide in a number of cases and they say that most of the cases responded well and that aluminium hydroxide offers many advantages over the older antacids. They consider that it is most effective when taken shortly before eating. They think that frequent small feedings are an undesirable stimulus to gastric secretion. They also think that while giving this drug a much more generous and less troublesome diet can be given.

It should be remembered that the dose of an alkali is more important than its choice. The dose should be sufficient to ensure continuous neutralization. Seven or eight doses may be necessary for a day. The higher the acidity of the stomach, the larger the dose should be. At the same time alkalosis should be avoided. One should steer clear of both the devil and the deep sea.

Alkalies are best taken from half an hour to one hour after meals but frequently they must be given at other times, whenever signs of hyperacidity are present.

Alkalosis is said to be an occasional complication of the intensive alkaline treatment. It is dangerous only when it is not promptly recognized. It is characterized by increase in the amount of bicarbonate or hydroxylions in the blood plasma. Besides overdozing with alkalies, alkalosis may be due to high intestinal or pyloric obstruction etc. The early symptoms are lassitude, loss of appetite, headache, depression, intense thirst and vomiting. Treatment is easy and simple when once the condition is recognized. All that is necessary is to stop giving the alkalies. If symptoms persist prescribe ammon. Chloride gr. XV T. I. D. or acid sod-phos gr. XXX t. i. d. Ammon. Chloride may cause vomiting. So it is better to give it per rectum (10 ozs. of 2 per cent sol.) and repeat, if necessary.

Special drugs :—

Of Special drugs for inhibiting secretion the most valuable is Belladonna and its alkaloid Atropine. Tr. Belladonna is given in doses of from 5 to 15 minims three-quarters to one hour after a meal. Some authors prefer the use of solution of Atropine, regulating the dose from one-two hundreth to one-hundreth of a grain. If the patient is sensitive to atropine he would have unusual and uncomfortable thirst. According to Whitla a convenient and efficient method of administering atropine is to place a tablet of $\frac{1}{200}$ gr. under the tongue, first thing in the morning and the last thing at night. The dose may gradually be increased to $\frac{1}{150}$ and $\frac{1}{100}$ gr. if there are no signs of intolerance, and tachycordia, excessive dryness of the mouth. At times belladonna is combined with bromide but it is likely to cause irritation in the stomach. If carefully used, belladonna is exceedingly useful. As a general rule, belladonna and atropine are not necessary in ordinary functional hyperacidity. It is said that doses of belladonna that reduce acid secretion also reduce secretion of protective mucin. In the usual doses given orally it is practically without effect on gastric secretory and motor functions (Eusterman.)

Moore states that large amounts of alkali may be definitely harmful as urinary calculi may arise from their use. Further he says that the urine of these patients becomes thick with phosphatic deposits and phosphatic calculi may develop.

njection of progynon :—

Schulz conducted some experiments recently with female ex hormone in ulcers of the stomach and intestine. 10,000 units were injected every second day. Latter it was reduced to 1000 and 500 units. After 8 to 14 days of treatment, gastroscopy showed that the ulcers had disappeared. The author recommends fractional injection of small doses. It is specially effective in ulcers caused by spasm of the capillaries as it probably produces its effect by a dilatation of the capillaries and hyperaemia of the gastric mucosa.

Posterior—Pituitary powder insufflation :—

Metzetial adopted this method of treatment in a number of cases, believing that the posterior lobe of the pituitary gland plays an important part in the eteology of peptic ulcer. Two-third grain was insufflated, four times a day for 5 or 6 weeks, then reduced to half the dose for 2 more weeks. Diet was not substantially modified, nor patients were confined to bed. Oral administration was ineffective. There was gain in appetite, weight and strength ; ulcer disappeared in a number of cases. This method should, however, be regarded as only complementary to the accepted principles of peptic ulcer management.

Histaminase :—

Hollaren reported a case of peptic ulcer having been treated with histamine. The patient was given 20 units of the drug 3 times daily before food. Ordinary diet was given. Alcohol and heavy smoking was allowed and yet he became symptom—free in 20 days. Then the dose was reduced by 10 units a day, until a maintenance dose of 10 units was reached. There has been no recurrence till now.

Histidin is employed as an isotonic sol of 4 % Histidin Hydrate of chloral in ulcers of the stomach and intestines. Daily ims. or iv. injs. of 5 C. C. are given for 20 to 24 days. The immediate response is uniform and prompt, but the improvement seems to be symptomatic and transcient.

K. A. Martin of Cornell University considers that the extravagant claims made for histidin are unwarranted.

Larostidin is an isotonic sterile 4% sol. of 1—histidin—mono hydrochloride. Intramuscular injections are given daily for about three weeks. Box of 6 and 25 amp, 5 c. c. each is available. Roger Korlexh of Oberhansen, Germany, considers it fortunate that Weiss and Aron introduced the use of larostidin in the treatment of gastric and duodenal ulcers. Some investigators found that it not only produced healing without decreasing the emptying time of the stomach, but decreased abnormal gastric secretion. It had a favourable effect on gastritis. Korbsch thinks that the results with larostidin are amazing. Mild and hypertrophic gastritis respond rapidly to this treatment. Deep, acute and chronic ulcers become flat and heal completely. Larostidin has been regarded as a great contribution to internal medicine. Martin, however, thinks that, though every now and then remarkable results of variable duration are obtained by the use of larostidin, one sees equally as many failures. *It should not be given if blood-pressure is high.*

Acids in gastric disease

When there is a defective secretion of hydrochloric acid, the natural inference should be to supply this deficiency by acid given by mouth. In some cases both pepsin and Hcl is absent from gastric contents and in such cases the addition of acid could not be of much service. In some cases, however, pepsin is present in achlorhydria and no Hcl. Yet after the addition of a suitable amount of this acid, digestion proceeds quite rapidly. Boas claims that acids are practically of no use in aiding digestion and that any influence that there may exist is on the motility of the stomach. Evald suggests the use of. 90 to 100 drops of the dilute acid. Sometimes it is given with pepsin and Tr gentian co. The dilute acid, in doses of 30 ms., is best given in half a tumbler of orange juice sherbet which should be sipped every now and then through a straw to save the teeth while taking a meal.

Some patients with Achylia cannot tolerate acids at all and do much better on alkalies.

Stomachics

Bitters and aromatics (gentian, bitter orange, lemon, calumba, cheretta, garlic etc.) are supposed to increase gastric secretion and therefore are used in cases of subacidity. Except in combination with general tonics, these are not of much value in the treatment of gastric disease.

Vitamin requirements

To provide for vitamin requirements for these patients it is now considered to be essential as free choice of foods is restricted. Various vitamin concentrates are handy preparations. Porturoy and Wilkinson investigated the blood content and urinary excretion of Vitamin (ascorbic acid) in patients with peptic ulceration and haematemisis and found that they suffered from severe vitamin C dificiency. Large doses of Vitamin C should therefore be given to all patients of peptic ulceration and haematemisis. Orange juice, strained, should be given from the very commencement of treatment. The adult daily requirement of vitamin C is about 500 units. The daily prophylactic dose is $\frac{2}{5}$ to $\frac{3}{4}$ gr. and the daily therapeutic dose is $1\frac{1}{2}$ to 4 grains. Ascorbic acid is generally given orally. It may also be given subcut, intrm. or intra-venously. Ampules containing 100 and 500 mg. are available. Cebion (merck), Redoxan or Redoxon Fort (Roche) may be used.

For Vitamin B, Benerva, berin, betaxan, collosol B, torulin or Vibex may be used. Capsules and tablets are used in the treatment of moderate deficiency. In urgent cases ampules should be used. Objon's Thiamate ampules also are very good. Williams recommends Marmite or Ryzamin B.

The A and D factors may be given in capsule form. One gram of Oleum Hippoglossi—Halibut liver oil—contains not less than 30,000 units of vitamin A with usually 2,500 to 3,500 units of vitamin D activity. Dose is 1 to 5 minims.

General measures

Tonics :—

The value of general treatment by tonics and iron in debilitated patients should be obvious. Treatment of the anaemic condition, slight as it may, is essential and iron preparations should be given at an early stage. The benefit of iron treatment is less marked when alkalies are being given. The progress should be noted by estimating the haemoglobin at intervals.

Laxatives :—

If the patient is taking Magnesium salt in suitable dosage, laxatives may not be required. A loaded colon means pyloric delay and gastric spasm. If necessary, the lower bowel should have saline washouts. Simple vegetable aperients are required at times to remove constipation, if little alkali is being used.

Rest in bed, in ulcer cases, should be the rule for the first fourteen days. After this the patient may sit up for short periods. Later on he may sit up daily to take one or two of his meals.

Alcohol and tobacco should be avoided in ulcer cases.

Foci of infection :—

The important part played by infective factor can scarcely be doubted. Some time ago Rosenow proved by experiment that both acute and chronic ulcers could be produced by injecting streptococci taken from septic throat and mouth. "A blood borne infection," says Whitla, "may also account for some cases that arise after acute infection, but a more important approach is probably by the swallowing of infected material direct into the stomach." All sources of infection should therefore be eradicated in every case of ulcer. The most important sources from oral sepsis are, pyorrhoea, periodontitis, and apical abscesses. Indeed pharynx and accessory nasal sinuses, septic tonsils, and chronic nasal cattarh are also responsible.

Recent observations have shown that it is always necessary to examine the prostate of men with ulcer.

Examine the urine for leucocytes and streptococci before and after prostatic massage as latent chronic infection may be lurking. If so, treat it with massage and autogenous vaccines.

Electric thrapy, massage and abdominal exercises may be useful in certain digestive ailments.

The Chief Complications of gastric and duodenal ulcer and their treatment

1. Haemorrhage :—

This is a late and serious complication. It is considered to be the most common complication of ulcer (Hurst and Ryle.). According to some clinicians bleeding occurs in about 25 per cent of cases. Haematemisis may be due either to gastric or duodenal ulcer, the latter is more apt to lose blood by the bowel (Melaena) when faintness or syncope will be the chief symptom with pallor and nausea. Later on tarry stools are passed. Other causes of alimentary bleeding must be diagnosed and excluded.

Medical opinion to-day is divided into two diametrically opposite camps as to the most suitable means of treating not only the severe but also the mild case of gastric bleeding. Hurst, Ryle, Witts and some others form one group. The leaders of medical opinion in Britain hold the opposite view. Morphia, hypodermically, restriction of both food and fluid by the mouth, rectal salines followed by graduated feedings are advocated by Hurst and some others; whilst Menlengracht and his followers recommend immediate feeding. Adequate food and fluid are given from the first day. The feeding method, however, does not seem to be suitable for every case. Generally the patient finds it almost impossible to take feeds at all during the first day or two.

The Starvation method :—

Morphia $\frac{1}{4}$ gr. is immediately given and repeated as required. The foot of the bed should be raised when the patient is found to be quiet and comfortable; the lower colon should be gently washed out with normal saline. Saline containing $7\frac{1}{2}$ per cent of glucose should then be given rectally by instilling 6 to 8 ozs., six hourly. The continuous drip method may be used instead. Nothing is given by the mouth for a day or two. After this period small feeds of citrated milk and Benger's food are given. Bye and bye, the feeds are increased. Generally these patients with bleeding are free from symptoms of dyspepsia and it is rarely necessary to give alkali.

A blood count and haemoglobin estimation are advised as soon as the patient is under treatment and the blood group recorded. A donar should be in readiness. A haemoglobin below 30 or 40 per cent. indicates the need for transfusion.

Hurst advocates the following line of treatment in haemorrhage. Immobility of the patient is necessary. Sufficient morphia by repeated subcutaneous injections are given to keep him drowsy and to prevent the physical and mental restlessness which are likely to be present. Atropine should be added to the morphia in order to inhibit the secretion of gastric juice ($\frac{1}{8}$ gr. morph Hcl with $\frac{1}{100}$ gr. atropine sulph.) Immobility of the stomach and duodenum is secured by giving the patient neither food nor drink by mouth. Complete starvation should be continued until, so far as can be ascertained, no bleeding has occurred for 48 hours. The ordinary strict treatment of an ulcer with diet, alkalies, atropine and olive oil should then be begun. During the period of starvation, the mouth should be kept scrupulously clean; there is then very little danger of parotitis. About 15 ozs. of 4 per cent dextrose sol. should be given by rectum alternately with 15 ozs. of normal saline sol. every 6 hours. A daily enema should also be given as the dextrose and saline solutions are then more likely to be retained. If mouth is parched, saline sol. should be given subcutaneously. 'To the stomach' says Hurst blood is as much a food as any other protein so that as much gastric juice is secreted and peristalsis occurs as actively when it is filled with n ordinary meal. It is common knowledge that uterine aemorrhage can almost be arrested by emptying the uterus and causing it to contract. The same principle holds good in this condition as well.'

A stomach tube is passed just far enough for the end to reach only the cardia. The stomach should be emptied with a senoran's evacuator and is washed out with about ozs. of ice-cold water and the lavage is repeated over and over until the water which comes back is no longer blood-stained. Instead of water, 1 in 1000 ferric chloride sol., also iced, can be used. When the last traces of water or ferric chloride sol. have been evacuated a drachm of 1 in 1000 adrenaline chloride is poured into the stomach before withdrawing the tube. The complete evacuation of the stomach and the stimulation of the mucus membrane with ice-cold water result in firm contraction of the whole organ and the haemorrhage is almost invariably arrested if it is gastric in origin. Duodenal haemorrhage also generally stops. The gastric juice should be rendered inactive by neutralizing as completely as possible all the free Hcl. which is present. Besides atropine, Tribasic phos. of Magnesia in drachm doses should be given, shaken up in a little water as soon as possible after the haemorrhage. Then it should be given every hour during the day and every two hours during the night unless the patient is asleep. This should be continued till feeding is begun. Afterwards usual treatment of ulcer is given. *Soda bicarb should not be given in haemorrhage.*

Whitla advises intra-muscular injection of calcium gluconate, horse serum, human blood and proprietary preparations of Haemostatic serum. The best, in his opinion, is the injection into the buttock of 10 c. c. of healthy human blood taken direct from the vein of a donar.

Whitla says that Menlengracht has reported very favourable results from abandoning the usual starvation treatment of Haematemisis and substituting for it a full diet which is commenced as soon as the patient is seen, however, severe the condition. About six meals a day are given, the food being soft such as porridge minced meat etc.

At any moment, a practitioner may be called upon to treat a case of haemorrhage. There are very few conditions in which life and death rest so directly upon the treatment one adopts. Every practitioner should possess a stomach tube with senoran's evacuator. Besides sphygmanometer, Haemoglobinometer should also be had. This should not be regarded as only a specialists' toy which no general practitioner save a haemotologist is expected to handle. Every district should have one man who can group a specimen of blood and who can carry out a transfusion of blood. As it is in the big cities of Britain, there should be a list of donors, properly grouped, who should be available at a moment's notice in emergencies. I am glad a blood bank has come in existence at Guindy.

2. Perforation :—

Though numerous lives are now-a-days saved by early operation, perforation is still much the most common cause of death. It may occur at any time in the history of ulcer but it occurs nearly always in chronic ulcerations.

Position of ulcer :—

More than 90 per cent of chronic gastric ulcers occur on or near the lesser curvature, an inch or more from the pyloric sphincter. A majority of them are found at least two inches distant from this point. Perforation takes place most frequently in ulcers in the pyloric and prepyloric regions. When it occurs, it is usually a rapid process even in chronic ulcers. Sudden sloughing is likely to take place. Perforation happens most frequently between one and two hours after a meal. It may take place immediately after straining at stools, during coughing or sneezing or as a result of vigorous exercise of riding etc. The pain starts quite suddenly and reaches a climax of violence.

Diagnosis of perforation :—

There is no condition in which immediate and correct diagnosis is of greater importance as every hour's delay in operating reduces the chances of recovery. The sudden onset of violent abdominal pain and the rigidity and tenderness point to this emergency. The diagnosis is confirmed by radiological investigation.

Treatment of perforation :—

Unless an operation is done at the earliest hour possible, it is almost inevitably fatal. The result of operation depends upon the interval between the perforation and the operation which should not be deferred until peritonitis sets in which may be local or general. After free exposure, the perforation should be closed.

After-care of ulcer cases :—

The importance of this aspect cannot be over-estimated. Mere freedom from symptoms is not enough. An x-ray examination should be done after six weeks to find out whether the ulcer has healed up. For further proof, faeces also should be examined for occult blood. For a long time after, the diet should be nourishing but simple and non-irritating. Short and regular intervals should be observed in taking food. All hardly digestible or indigestible articles and sour or highly seasoned foods should be scrupulously avoided. Chill and over-fatigue physical and mental, are harmful. Alcohol and smoking should be prohibited. Hygiene of the mouth should be attended to. One should not go without food for longer than 2 or 3 hours. The antacid should be continued regularly to keep the hyperacidity in check. Anxiety and depressing emotions should be discarded.

Cushing relates ulcer formation to abnormal parasympathetic discharges and vagotonia. Vagus stimulates peristalsis. Beattie recently demonstrated that hyperchlorhydria, haemorrhage and ulcer formation may result from stimulation of the tuber cinereum. 'It seems probable,' says Whitla 'that the dual nerve-supply to the stomach, the craneo-sacral automatic system and the thoraco-lumbar sympathetic system represent a balanced and mutually antagonistic mechanism so that highly strung persons who incline to that form of nervous instability called vagotonia are particularly prone to chronic hyperchlorhydria and increased gastric peristalsis often leading to ulcer.'

Dietetic Treatment

"Diet cures more than the lancet"

"Diet is the basis of treatment and milk is the foundation of diet." In all digestive disturbances, diet plays an extremely important role in their treatment. In most of these affections cures can often be effected by diet alone but without this mode of treatment, serious complications are likely to arise. Therefore every practitioner should fully grasp the principles that govern the use of food.

"**What is the chief object of dietary regulation** in diseases of the digestive organs? Is it to spare these organs as much as possible and thus to permit them through rest to recuperate? In some instances such a procedure is good, in others it is bad (the old adage 'லங்கணம் பரம ஔஷதம்' does not hold good in all cases). In acute gastritis, in gastric ulcer and in acute diarrhoea, for instance, rest of the digestive tract is obviously needed. Fluids, chiefly milk and semisolid foods are given. On the other hand in mild chronic gastritis with subacidity and particularly in functional disorders of psychic origin too much protective therapy may work harm. The old starvation treatment of digestive diseases is fortunately now a thing of the past and it is generally recognized that it is difficult to cure a patient of any disease (except perhaps acute gastritis) by prolonged starvation (Mclester).

In prescribing diet in digestive disorders three essential factors must be borne in mind; the food must be in the most digestible form so that the work of the stomach is reduced as much as possible; second, its bulk should be small and third, its character must be rich as to increase the patients' nutrition.

The essential role of the stomach is a mechanical one rather than truly digestive. Therefore the physical form of the food rather than its chemical composition, must be considered as important in dyspepsia. The first and foremost rule to be observed in disorders of the stomach is to see that the form of the food is such that the stomach finds little or no difficulty in driving it on into the duodenum. Therefore it should be in a fine state of division and must be carefully chewed and masticated. "Well chewed is half disgested."

Then comes the question of bulk. The larger the quantity the greater is the muscular labour imposed on the stomach. Animal foods, are as a class, less troublesome to dyspeptics than vegetable products. The meals of a dyspeptic, therefore, should be of small size, taken at rather short intervals.

The character of diet, besides being highly and sufficiently nutricious, must be considered from the standpoint of gastric secretion whether the condition present is hyperacidity or subacidity. In hyperacidity an abundant proteid diet should be advised in order to neutralize the excess of hydrochloric acid but strangely enough some patients at times do better on bread and starchy food. In such cases carbohydrates must be allowed only in moderate amounts in a most digestible form. In subacidity proteins are not easily digestible but the carbohydrates are digested as a rule without difficulty.

Liquid Food in Gastric disorders :—

If you prescribe liquids in order to spare the stomach, milk is the article which is usually best borne. It is best taken diluted with either lime water or Vichy. Sodium citrate renders it more digestible. To enhance its palatability, a little vanilla essence may be added. In cases in which it is badly borne, butter-milk, acidophilus milk, kefir or kumiss may be used instead. Tea and coffee are better avoided or if at all only weak infusions should be taken. Cocoa is highly nutricious and is a valuable liquid food for patients of peptic ulcer. Meat juices are decidedly nutricious but should not be given in hyperacidity or gastric ulceration.

Light and bland forms of food are allowed. Soft boiled eggs are most digestible. Raw eggs pass the stomach unabsorbed. At times poached or scrambled eggs are preferable. Fish containing little amount of fat should be prefered. The usual form of meat may be rendered more digestible by scraping and grinding.

Vegetables should be given cautiously if there is marked fermentation. Puree is the best form of many a vegetable. Cabbage does not generally agree with patients suffering from gastric affections. So also root vegetables, lentils, pulses and dhals. Potatoes are most easily digested in baked or mashed forms. Fat meats and greasy gravies should be avoided.

Special factors relating to the diet of patients of gastric disorders :—

When the gastric secretion fails, the intestines vicariously perform the work of the stomach. When there is severe vomiting or when food is not digested, nourishment may be had through the channels other than stomach as by rectal feeding or direct duodenal feeding with the aid of duodenal tube.

The food should be thoroughly masticated not only to divide it mechanically but to increase salivery secretion which in turn has an important influence on the digestive functions. The temperature of the food is also of paramount importance. It should be between 98° and 100° F. Very hot or cold food is injurious. The question of rest and exercise is of great importance in gastric disturbances. In superacidity and subacidity rest after meals should be ordered but in patients with motor disturbance of the stomach, moderate exercise should be advised. Rest, violent exercise or sleep soon after meals alike disturb the digestion in this condition. Meals should be taken at regular intervals.

Milk cure :—

"Diet is the basis of treatment and milk is the foundaron of diet." This consists in the administration of fairly good amounts of milk, given alone or mixed with other foods. It is more easily digested if given with starchy foods such as rice, sago or barley. It is of special value in ulcer of the stomach and some forms of chronic gastritis. It may be enriched by the addition of cream in gastric ulcer. Gratifying results have been observed in certain forms of gastric neurasthenia. All the same in some patients or in some conditions it disturbs the stomach and causes flatulence and under those circumstances it should not be employed. It is especially badly borne in subacidity and in Achylia gastrica. It should be contra-indicated in servere types of atony and dilatation as well as in intestinal disturbances with marked flatulence and in most forms of chronic diarrhoea. Milk may produce constipation which can be counteracted by the addition of milk or magnesia.

Fat :—

In many cases fat is apt to cause trouble. This is particularly true of cooked fat as in process of cooking it liberates fatty acids, and other irritating substances. On the other hand butter—fresh cow's butter, does not cause difficulty to that extent and it is beneficial in ulcers of the stomach.

Fat may be good in certain cases of hyperchlorhydria, but it is injurious in atonic dyspepsia accompanied by marked dilatation. Olive oil, butter and cream are the chief substances used in gastric disease (1 to 2 ozs. of butter or 1 oz. of salad oil taken before meals) and often prove of great service in counteracting the acidity so frequently present in duodenal ulcer (Mclester). Olive oil taken before food diminishes the secretion and increases the influx of bile and has the further advantage of supplying a digestible and absolutely un-irritating food of very high nutritive value in a concentrated form. Half an ounce yields 144 calories. Half to one ounce may be given every day before all the 3 or 4 meals.

Diet in gastric and duodenal Ulcer

Certain articles of diet are of special value. **Milk** comes first and foremost. It should be the sole food in the beginning and even as the treatment proceeds it should be the mainstay of the diet. For many years after the cure is established this should be an important adjunct to the new food.

Cream may be added to the milk in varying proportions, say up to one half the volume.

Eggs rank next to milk. Half boiled ones are the best. Three to six per day may be allowed. Cooked cereals occupy the third rank: Boiled rice, sago, oatmeal farina, cream of wheat. The diet should contain all the essential nutritive factors and it should be a well-balanced one and its vitamin content should not be lost sight of.

Meat and meat extracts :—

The stimulating influence of meat and meat extracts, is often of benefit in the treatment of digestive ailments but not in hyperchlorhydria and peptic ulcers.

Oils and fats :—

All recent work tends to confirm the fact that oil diminishes gastric secretion. Oils and fats also act by causing some regurgitation of alkaline juice from the pancreas. It is a well-known chemical fact that oil has a much more beneficial effect when taken on empty stomach before meals. When taken after food it may do no good. Oils and fats have certainly a tendency to retard emptying of the stomach and if this is to be avoided, cut off all food by the mouth and rectal feeding should be resorted to for even 2 or 3 weeks at a time if it be so necessary. Gradually, suitable diet should be allowed. Any recrudescence of symptoms must be the signal for immediately retracing one's steps in reducing the diet. Very cold and very hot articles of food should also be avoided.

The guiding principles of the **Lenharty diet** are hese :—

(1) To promote healing of the ulcer by giving as much nourishing food as possible. (2) to 'fix' the acid of the gastric juice, and so to prevent its interfering with healing by ensuring that the food contains a large proportion of protein ; (3) to prevent distension of the stomach by the use of small feeds. (Hutchinson.)

The diet should produce as little secretion of acid s possible or render it inoccuous by combining with it. egetable purees can be cooked with salt but it is not dvisable to add more salt. Hurst advises not to give nything containing chlorophyl as this would interfere ith the examination of faeces for blood etc., milk toast, ashed potatoes, strained juices of orange and other fruits re permissible in the later weeks of treatment. In the cale of permitted foods the following articles come last— ender meats, fish, once a day, puree vegetables and he simple rice or tapioca puddings or milk custards.

The following foods should be prohibited :—

Broths and meat extracts, dried fish, smoked or dried eats, coarse and woody vegetables, raw fruits, highly easoned sauces and foods pickles, all alcoholic drinks nd tobacco smoking and chewing.

Alkali therapy is intimately related to the dietary egulation of ulcer. *Sippy's treatment* is considered to be oo elaborate for the general practitioner and his poor nd ignorant patients.

As much as possible *rest* for the organ is the point chiefly to be aimed at arranging a diet for ulcer cases. est means the protection of the mucus membrane from oth the mechanical and chemical irritants. In very evere cases, especially, if there has been recent bleeding, bsolute rest is needed. One or two feeds should be kept y the side of the patient so that he may have the ame when awake during night.

Diet in Acute Gastritis

The dietetic treatment of *acute gastritis* should proceed on the same lines as that of gastric ulcer. Since the acute form is of short duration, rectal feeding is rarely required. Food should be withheld until vomiting ceases for 24 to 48 hours. The diet should be planned to avoid all mechanical, chemical or thermal irritation of the gastric mucus membrane. During the starvation days, boiled water, thin barley water or albumen water are given. When the acute symptom abate, liquid feeds are given for a day. Milk and soda, citrated milk (sod. cit. 5 grs. to 1 oz. of milk) milk flavoured with tea, Benger's Food or Allanburys food may be given. If all goes well, the next day, diet may be improved and increased. By the end of a week it may be possible to resume ordinary diet.

Simple Chronic Gastritis :—

The common causes are habitual consumption of indigestible food, imperfect mastication, continued over-indulgence in alcohol, tobacco, condiments, sauces, pickles, drugs etc. (Whitla.)

The treatment consists in the removal of the offending cause. Regularity of meal-times, moderation in diet should be advised; only a bland non-irritating diet should be allowed. Dietetic directions are the same as for the after-care of gastric and duodenal ulcers.

Diet in Chronic Hyperchlorhydric Gastritis or Acid Gastritis

This condition is believed to be the precursor of gastric ulceration. In this condition there is hypersecretion and an abnormal rapid gastric motility; consequently the stomach gets empty soon and is under the influence of irritating gastric juice for greater part of the day than in normal subjects. It is, therefore, more liable to suffer by alcohol, tobacco, drugs etc., which set up chronic gastritis.

Avoid the use of anything that may irritate the gastric mucus membrane or excite a secretion of mucus.

ustard, pepper, spices, condiments, fried fats and pickles hould be forbidden. So also alcohol and tobacco. offee is apt to be harmful owing to the oily substances hich it contains, sugar, especially cane-sugar, is very nsuitable as it is irritating. Butter in moderation is greeable. Toasted bread is allowed, Cauliflower is the ost easily digestible of vegetables.

iet in Chronic Achlorhydria Gastritis :—

The test meal gives some indication of the severity the gastritis. In advanced cases in which atrophy of the ucus membrane has occurred and involve the superficial ucus secreting cells as well as the tubules, mucus is bsent and total acidity is much reduced.

Dry food to stimulate saliva is given. Bulky meals hould be avoided. Food should be easily digestible nd non-irritating in character. Liquids should be taken part from meals or between meals; soups and meat xtracts are valuable stimulants of gastric juice. These hould be taken before meals. A puree of spinach பசலிக்கீரை) is particularly useful as it contains a substance ke histamine which is a good stimulant of gastric juice. he patient should rest half an hour before and half an our after a meal. If any mucus is found in the test meal he stomach should be washed with dilute hydrogen eroxide (1 to 4 drs. to 1 pint). The test meal should e repeated even after the mucus has disappeared. ll causes of gastritis should be removed wherever ossible. In achlohydria the excess of mucus is a poor ubstitute for gastric juice and the consequent loss of the econd line of defence leads to irritation of the small ntestine, so that duodenitis and enteritis develop in ddition to the gastritis (Price.)

Diet in Atonic or Flatulent Dyspepsia (Defective Motility)

In this the mechanical form of the food is of chief nportance. The stomach should not be burdened with large uantities of material especially with bulky vegetables. olids and liquids should not be taken together. Fluids should be taken apart from meals, preferably between meals. If there be flatulence, pulses, lentils and vegetables with 'windy' tendency should be avoided. Starches should be limited. Tea and coffee, if taken at all, should be weak as these cause flatulence. In atonic dyspepsia, it is said, that alcoholic beverages (a little sound wine) give happy results as alcohol is a stimulant both to the secretory and motor functions of debilitated stomach. Spinach and cauliflower tops are allowed. Woody and stringy vegetables should be avoided. So also root vegetables are to be discarded. Potatoes only sparingly to be had, crisp toast, and rusks should be taken instead of ordinary bread. Sugar and jam and pastry should not be indulged. These rules are applicable in all cases of moderate degree. In severe cases, an exclusive protein diet may be indicated. In this, fluids are badly borne and therefore they are taken between meals.

Diet in Nervous Dyspepsia

Most neurasthenic patients suffer from this form of indigestion partly from the abnormal irritability of their nervous system and partly as a result of chronic toxaemia, worry, excitement, anxiety and other depressing emotions, and overwork, physical and mental. These tend to inhibit the motor and secretory functions of the stomach. First of all the patient should be assured that he has no organic disease. The general neurasthenic condition requires first attention. For this both, mental and physical rest, exercise, graduated of course, and sufficient and nourishing food to overcome the inanition are the prime indications. Congenial surroundings, brazing cilmate, pleasant companions and appetising food play a great part. The general state of neutrition should be improved at all costs.

In this condition, "it is often necessary to throw dietetic rules to the winds and to feed the patient up."—Hutchinson. Nervine tonics, and psychotherapy play most important roll. "So long as the patient is being treated, he is also being examined; so long as he is being examined he is also being treated"—Ross.

Individualization

In the dietetic treatment it is important as Hutchinson points out, to individualize and adapt the diet not only o the patient with due consideration of his likes, dislikes and idiosyncrasies, but also the particular disorder of unction.

Easily digestible food-stuffs

The following articles of food are comparatively the most digestive and make least demand upon the secretory and motor powers of the stomach.

Meats :—Mutton, sweetbread, young chicken, tripe, rabbit, partridges and certain birds.

Fish :—Whiting (களங்கான்), sole, turbot (steamed or boiled).

Starch Foods :—Stale toasted bread, rusks, rice, sago, arrowroot.

Vegetables :—Asparagus, seakale, spinach (பசலி), snake gourd (புடலங்காய்), young brinjal, cauliflower, French beans.

Fruits :—The juice of orange, of grapes and of pomegranates, baked apples.

'Made dishes', twice cooked meats, sauces, pastry, cakes, pickles, cheese, sweets, jams, jellies, preserves should be avoided. Meat should be underdone. Ghee instead of oils should be used in cooking foods.

Four cardinal rules regarding diet in gastric disease

(1) The patient should avoid any article of food that tends to cause physical irritation in the stomach (such as raw and coarse vegetables, currants, raisins, nuts with indigestible residue).

(2) Meal which tends to cause a marked secretion of gastric juice should be avoided in diseases like Hyperacidity and ulcers. Beaf, tea and meat extracts should especially be avoided.

(3) Great care should be taken, never to take too large a meal at one time.

(4) The food should be thoroughly masticated so that it enters the stomach in a homogenous mass.

Modern diagnostic methods and appliances.

Gastroscopy :—

It is said that 'gastroscopy' is as essential in modern clinic as cystoscopy but it is not generally available to the profession at large. Whitla says that in the hands of experts this method in sometimes of value in detecting ulcer or early gastric cancer. By some clinicians it is considered as the best diagnostic procedure. Cicatrization of septic ulcers can be observed only by gastroscopy. If neither ulcer nor cancer can be demonstrated roentgenologically, its use is indicated. The flexible instrument, improved by Heinning and Wolf is the most common in use. If it is not possible to introduce the soft tube, it is contra indicated. In the opinion of Pearse Williams, gastroscopy is as yet too young an art to be considered as a routine method of examining the stomach. According to Hurst its chief value is in the recognition of the different forms of gastritis and of erosions and acute ulcers, which may cause haemorrhage but are never recognisable with x-rays. It is often helpful in diagnosing the cause of post operative symptoms when radiology has failed. It is not of much use in diagnosing early cancer.

The Fractional test meal :—

This test gives information about the average level of acidity, the persistence of hypersecretion, the rate of emptying and the presence or no of stasis. Whitla says that the test is of chief value in detecting achlorhydria and in confirming the presence of hyperchlorhydria in suspected ulcer. It also gives valuable information of the emptying of the stomach and in cases showing retention with x-rays may be used as a control test to decide whether medicinal or surgical treatment is indicated. The test is performed in the morning on a fasting, omach, the patient is made to swallow a fine rubber be (Ryle) and the fasting gastric contents are aspirated efore the test meal is given. The meal consists of fine ruel. Samples of gastric contents are aspirated at intervals r two to three hours and examined for blood, bile, ucus and free H.C.l.

This test is not used as frequently as formerly. "Recent ork" say Todd and Sanford "has thrown much doubt pon the value of the fractional method as originally arried out." Maclean considers it to be too time consum- ng and the charges for such an examination even in a Government laboratory are too heavy for a patient of verage means. Nor is the additional information which gives of sufficient value to compensate its costliness.

he Barium Meal :—

According to Whitla this is valuable in detection of lcers or infiltrating growths of the stomach or duodenum hen screening examination is made and radiographs aken at the different stages of filling and at different ngles of projection. The test also gives information of he rate of the emptying of the stomach, a six hour astric residue generally being accepted as evidence of etention. The patient receives the opaque meal in the norning; fasting and no special preparation is necessary. Opaque drugs such as bismuih should be stopped for or 3 days before the barium meal is given.

Radiography :—

Confirmation of the presence of gastric ulceration is sually obtained now-a-days by this method. Its prime alue is not for making the diagnosis but to confirm for nstance the presence of ulcer, to reveal its extent, to ecord the emptying time of the stomach and also to ontrol the degree of healing, obtainable by medicinal reatment.

Maclean thinks it altogether erroneous to consider hat X-rays constitute the only reliable method of diagnosis gastric disturbances. A correct diagnosis can be arrived without this aid. However X-rays are exceedingly useful in confirming our clinical findings or at times to cast doubt on them. The present day tendency to rely, entirely on X-ray examination is bad policy. 'It is not, says Maclean quite fair either to the patient or the radiologist.' "The best evidence of deficient motor power," says Todd 'is the detection of food in the stomach at a time when it should be empty, before breakfast in the morning.' A special test meal containing easily recognized materials (rice pudding with currants, jam with seeds, raisins or spinach) is sometimes given and removed at the end of six or seven hours. When more than 100 c. c. of fluid are obtained with the tube one hour after an Ewald breakfast, deficient motility may be inferred.

The Stomach Tube must be used with great care in suspected cases of gastric ulcer. It is even unwise to use the stomach tube, where ulcer is strongly suspected. The diagnosis should rest largely upon the clinical symptoms. The newer types of stomach tube especially the Rufuss tube, are to be recommended.

Examination of the faeces :

This test should not be neglected in cases of suspected ulceration or cancer. The faeces should be examined for occult blood, undigested fat and meat fibre. Black tarry stools indicate recent bleeding on a large scale. For doing the test for occult blood, the patient should be put for three days on a haemoglobin and chloroyhyl free diet which consists chiefly of bread, butter, milk, eggs, puddings and potato. Meat, fish, fowl, green vegetables and fruits are excluded.

In placing these views before you I cannot claim any originality. I have only played the roll of the honey-bee in culling and collecting the information from various sources, especially from the works of Maclean, Hurst, McLeister and Sir Robert Hutchinson.

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PRINTED AT THE CAXTON PRESS, MADRAS—1942.

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आगत संख्या 46,540E

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